TOBACCO AND HEALTH

The Indian Scene

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held at Bombay India, April 15-16, 1987.
Sponsored by International Union Against Cancer (UICC).

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FOREWORD

The International Union Against Cancer (UICC) had sponsored a Workshop on "Tobacco or Health" at the Tata Memorial Centre in April, 1987. I am glad that the proceedings are being published. The Tata Memorial Centre has done pioneering work in the field of tobacco and cancer and has continued to provide national leadership in this field. The Workshop covered not only the health hazards of tobacco in relation to cancer but also discussed cardiovascular and lung diseases where tobacco is a significant risk factor.

The Workshop has brought into focus the serious nature of this health hazard in the country which is estimated to take at least 600,000 adult lives every year. This menace will continue to grow in the future unless effective steps are taken to stem the tide. The proceedings bring together under one cover the available evidence from India about the burden of tobacco related diseases, the nature and extent of toxic chemicals in Indian tobacco and tobacco products. The findings reviewed in the volume bring out clearly that the main focus of intervention in India should be bidi which is at least as toxic as cigarette and is four to five times more commonly smoked. The scientific efforts that have been made to modify tobacco related behaviour in India are reviewed and the action programmes undertaken by the governmental and voluntary agencies have been highlighted.

The proceedings will be useful to (i) scientists for further work in the field, (ii) to media, which have a major responsibility to create awareness, (iii) to central and state governments for generating effective tobacco control strategies and (iv) to voluntary organizations to act as catalyst in a major area of health concern in the coming years.

Dr. M.R. SRINIVASAN
Chairman, Atomic Energy Commission and
Chairman, Governing Council, Tata Memorial Centre.

PREFACE

Studies on Tobacco is a priority research area at the Tata Memorial Centre. The National Cancer Control Programme, adopted by the Government of India during the Seventh Five Year Plan, has also given a high priority to prevention of tobacco related cancers. Interesting scientific data is now emerging in this area.

The late Dr. V.R. Khanolkar, the first Director of Indian Cancer Research Centre laid foundations for epidemiological and experimental research relating to tobacco chewing and oral cancer. Rural populations were studied with special reference to precancerous lesions. The important observation on the role of bidi smoking in relation to common cancers of the head and neck was made by Sanghvi and his colleagues in 1955 and was subsequently supported by his findings of high levels of tar, nicotine and carbon monoxide in the bidi smoke. Lung cancer became relatively common during the 60's and the 70's at the Tata Memorial Hospital and was shown to be related to bidi as well as cigarette smoking habits. Long-term cohort studies carried out during the 70's and 80's confirmed many of these findings. The studies during the 80's demonstrated (i) the role of bidi and cigarette smoking in heart diseases, (ii) the presence of new carcinogens in chewing tobacco and (iii) protective effect of certain diets on tobacco risks. Many of these studies have been confirmed by independent work carried out in different parts of the country.

It was, therefore, appropriate that the International Union Against Cancer (UICC) agreed to hold a Workshop on "Tobacco or Health" at the Tata Memorial Centre. This publication will be the first comprehensive document covering the available scientific information in the country on the issues of ill-effects of tobacco use on health. The area of enquiry has extended beyond the medical fields into education and behavioural sciences providing a body of knowledge on which the efforts to control tobacco related deaths and disability could be built. The sobering facts regarding tobacco economics, however, have to be kept in their proper perspective.

Tobacco may well be considered and indeed is the largest single preventable cause of death and disease; for a developing country like India, it would therefore be appropriate to devise ways and means to control this menace. The Tata Memorial Centre has always emphasised the great merit of this area in scientific research and we are committed to the field of Preventive Oncology in the years ahead.

Dr. P.B. DESAI
Director, Tata Memorial Centre

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Introduction

Scientific work carried out in India in relation to tobacco-related diseases has made it abundantly clear that the problem in the country is already as serious as in the western countries, although somewhat of a different nature. India is the third largest producer of tobacco after China and U.S.A. Although per capita consumption of tobacco in India is low, the manner in which tobacco is consumed, increases the health hazards considerably. For instance, from every gram of tobacco on an average, one cigarette is produced but three to four bidis.* The tendu leaf which is used as a wrapper in bidi, contributes more than half of its weight. Even with this limited amount of tobacco and its small size, every bidi delivers to an Indian smoker at least as much tar and nicotine as an Indian cigarette and much more of carbon monoxide and some other toxic chemicals. Indian cigarette, in turn, delivers more of tar and nicotine compared to the cigarettes marketed in the west. Bidi smoking is currently the most common type of tobacco habit both in urban and rural India and for every cigarette smoker, there are almost four to five bidi smokers in the country.

With a view to deliberate on the ill-effects of tobacco on health with special reference to Indian tobacco habits and to consider steps to stem this oncoming epidemic, a Workshop was organized at the Tata Memorial Centre in Bombay in April, 1987 under the auspices of the International Union Against Cancer (UICC). This Workshop was the first concerted effort in the country to bring together, scientists (epidemiologists, basic and agricultural scientists), medical experts, policy makers (officials of state and central health departments), social workers (representatives of voluntary agencies) and the people in the media, on one platform, (i) to critically evaluate the available scientific evidence, discuss its strengths and limitations and attempt a synthesis to arrive at an estimate of overall hazard associated with the use of tobacco products in India and (ii) to evolve strategies for their control through short-term and long-term measures to reach the goal of non-tobacco using generation.

The present volume covers the deliberations of this group and includes some additional material in the Appendix, on possible substitutes of tobacco crop and alternate uses of bidi tobacco. A paper on chronic obstructive lung disease, which though not formally presented in the Workshop, is included for completeness of coverage of ill-effects of tobacco use.

* Bidi is an indigenous product which is smoked.

Burden of Tobacco Related Diseases and Deaths

The contents of this volume are divided into four sections. The first section covers the information on overall mortality due to tobacco use as well as available information on morbidity and mortality of specific tobacco-related diseases viz., cancer of certain sites, cardiovascular disease (CVD) and chronic obstructive lung disease (COLD). Limited data on the effects of tobacco use by pregnant mother on foetal health and pregnancy outcome is also reviewed.

The quality of data in most of the Indian publications on these topics was reliable and has provided the backbone for various assessments made here. However, there are serious gaps in our current knowledge to undertake a synthesis of this material and arrive at a detailed picture of the health hazards due to tobacco use in the country. By far the most useful information in this context was available for overall mortality risks due to tobacco usage in rural as well as urban areas of the country. A great deal of information was available about the diversity and extent of different tobacco habits in the country. Figures were also available on the amount of different types of tobacco that was released for consumption in the country. On the basis of this information, an estimate of the prevalence of common habits in India was arrived at. Among the diseases etiologically associated with tobacco use, the most reliable information on morbidity was available for cancer. This incidence data, however, was available mainly for urban centres. Information on the prevalence of other conditions considered viz., cardiovascular disease, and chronic obstructive lung disease was rather limited. Information on mortality by cause of death was even more limited and had largely to be extrapolated from mortality statistics of Bombay City with necessary modifications, which are discussed in the text. Similarly, reliable information on quantification of risks associated with tobacco chewing and smoking habits for developing these diseases was available from several epidemiological studies carried out in different parts of the country but none was available for the risk of dying from these diseases.

Despite these limitations, on the basis of rational and conservative assumptions, a general picture of the health hazards due to tobacco usage in the country has emerged. Out of an estimated five million deaths in the adult population in the country, a minimum of 630,000 and a more reasonable estimate of one million deaths can be attributed to current use of tobacco in the country. As outlined above, a weak link in our further assessment is the information on the cause of death statistics in the country. Detailed examination of the available data strongly suggests that at least half of the deaths attributed to tobacco usage can be accounted for, by deaths due to cardiovascular disease, chronic obstructive lung disease and cancer of certain sites, which are also the major tobacco associated killers in the west. There are some differences in the nature of effects seen in India and in the west. For instance, cigarette smoking which is the most common habit in the west, leads primarily to lung cancer accounting for over 70% of tobacco-related cancer deaths and a third of all cancer deaths – in the US in particular. In India, where bidi smoking and tobacco chewing are the common habits, major effects of tobacco are seen in the oral cavity, pharynx, larynx and oesophagus

which together account for almost 75% of tobacco-related cancers that occur in the country. Overall, however, almost a third of all cancers involve sites which are related to tobacco habits.

Effects of poor nutritional status and inadequate health care of the Indian population would also be reflected in the mortality pattern. What we could not really assess was the role of tobacco in communicable diseases such as tuberculosis and pneumonia which are estimated to cause about a million deaths in the adults of the country. European literature after the Second World War had suggested an enhancing role of tobacco in the prognosis of tuberculosis and a more frequent recurrence of pneumonia. Since these diseases were controlled in the west by improved nutrition and sanitation and by therapeutic and other measures, no recent studies on tobacco risks associated with these conditions are available. These aspects,to our knowledge, have also not been studied in India.

There may be other health effects of indigenous tobacco practices which may turn up as in the case of the role of maternal tobacco chewing on foetal health and pregnancy outcome. In the west, current thinking has implicated carbon monoxide in cigarette smoke to be primarily responsible for these effects. Indian studies have demonstrated that tobacco chewing during pregnancy, where carbon monoxide is not involved, produces an effect very similar to cigarette smoking. These observations need to be confirmed.

In addition to these effects on mortality, it has been estimated that about 15 million cases of COLD, CVD and cancer of the upper alimentary and respiratory tracts prevalent in the adult population in the country are attributable to tobacco usage. A high proportion of this morbidity is accounted for by COLD cases as in the west, followed by CVD and cancer.

These estimates of health hazards of tobacco usage in India, although preliminary in nature, show the formidable dimension of the problem and suggest the areas in which urgent scientific work is necessary.

Reduction of Toxic Chemicals in Tobacco Products

The increasing onslaught of death and disease caused by the use of tobacco products can only be stemmed by developing appropriate strategies for control of tobacco use and implementing them vigorously. These aspects are covered in sections 2, 3 and 4. The National Cancer Control Programme which was launched in April, 1985 as a part of the Seventh Five Year Plan gives a high priority to eliminate tobacco-related cancers by the turn of the century. While long-term strategy should aim at eliminating all uses of tobacco, interim measures may be considered e.g. reduction in levels of toxic components in tobacco products. This aspect is covered in section 2. In this section, baseline information on the emission levels of certain toxic components like tar, nicotine, carbon monoxide, hydrogen cyanide, phenols and benzo(a)pyrene in the smoke of bidis and cigarettes presently marketed in the country, are reported. In addition, levels of carcinogenic agents, in particular, tobacco-specific nitrosamines in smokeless tobacco-mainly chewing tobacco and masheri (powdered burnt tobacco rubbed on

gums) – are also given.

Several experiments have been carried out to reduce the toxic components in bidi and cigarette smoke. Experimental bidis from tobacco plants with varying genotypes and grown with differing agricultural practices seem to have only marginal effect on the reduction of toxic chemicals in the smoke. Similar results are reported for cigarettes also. However, substantial reduction in the emission levels of toxic components of bidi smoke has been achieved experimentally by the use of cheap and effective filters or by other simple procedures. Reports from the west have shown that there is a lowering of risks of cancers of certain sites in those who smoke filter cigarettes, with lower levels of tar and nicotine, compared to smokers of nonfilter cigarettes. Efforts at reduction of toxic components therefore need to be pursued more vigorously so that tobacco companies in the country would be compelled to market less toxic, low tar-low nicotine bidis and cigarettes with specified upper limits (like some western countries where the upper limit for tar is 15 mg and for nicotine 1 mg, per cigarette).

The filter cigarette marketed in India is a misnomer, because filter king cigarettes have even higher levels of tar and nicotine than plain nonfilter cigarettes. Similarly filter bidis manufactured for export also have higher levels of tar and nicotine compared to long (75 – 80 mm) bidis. It needs to be reemphasized that these measures are to be considered as nothing but interim measures and as adjunct to primary prevention strategies to reach a goal of non-smoking generation. Relevance of low tar – low nicotine bidis/cigarettes under Indian conditions is discussed at length at the end of section 2. Research still needs to be undertaken to make smokeless tobacco less harmful.

Intervention Studies and Behaviour Modification

Understanding of the behavioural factors responsible for the establishment of tobacco habits and their maintenance over time are important for any intervention programme of modification of tobacco-habit behaviour. Though long-term benefits will depend on prevention of the onset of tobacco habits especially in children and adolescents, successful programmes concentrating on helping individuals to give up their habits, particularly in populations where they are already widespread, may provide immediate results. One such intervention study (section 3) was undertaken by a group at TIFR in rural populations of three states. Knowledge on the ill-effects of tobacco-use was imparted to 36,000 tobacco users through personal communication as well as mass media. Encouraging results with respect to change i.e. reduction or discontinuation of tobacco habits, have been reported and a concurrent significant decrease in the incidence of precancerous lesions has also been observed within five years of the institution of the programme. This is the only long-term study to have reached the stage at which impact of intervention can be observed. The other two studies covered in this section, undertaken in the state of Kerala by the Regional Cancer Centre and in the state of Karnataka by the Institute of Oncology (Bangalore), are at the stage of collection of baseline information i.e. prevalence of tobacco usage practices, and attitudes and awareness of health risks associated with these habits, which is needed to launch

intervention programmes.

One group which is particularly important for initiating preventive measures is that of children since they may not have as yet acquired the tobacco habit or may have done so only on experimental basis. A study of 1668 school children in Goa has reported that there was practically no smoking and very little chewing in children but over 50% of children who had tobacco habit, used masheri (powdered roasted tobacco). Probably it is used by elders in the family and hence easily available to the children. The micro-climate comprising of the family members, friends and teachers exert great influence on children and as mentioned in the paper by the Director of Education and Child Research Group of Cancer Research Campaign, UK, there is a need to make school teachers, parents and family members aware of the influence they wield with children and help them to be more responsive.

Strategies for Tobacco Control

Public information and public education programmes are the two essential components of tobacco control programmes on which action can be mounted by Central and State Governments, voluntary bodies like Consumer Guidance Society of India and others. These issues are covered in the last section of the volume. In India, till recently, the topic of tobacco control was mainly restricted to cigarette and did not touch bidi or chewing tobacco which are far more widely used in the country. This is now changing. As mentioned earlier, the Government of India has launched a National Cancer Control Programme which gives high priority to prevention of tobacco-related cancers by the turn of the century. The State of Maharashtra has taken a lead in this direction and appointed Working Groups to evolve State Policy. State activities have been outlined in a paper by the Director of the State Health Services which shows that steps have already been taken to make existing legislation operative, by prohibiting smoking and spitting in public places, all government buildings, offices etc. The ban is also extended to hospitals and hospital premises. Public awareness campaigns have also been instituted through newspaper articles, television and radio programmes, posters, hoardings etc. The nonsmoking zones of public transport, like state transport buses, are to be made more effective. Thus encouragingly, the state is committed to taking action within the limited resources available at its disposal.

Many pressures, particularly those of exemplar groups like that of medical professionals could be brought to bear on the relevant authorities. In most countries where action has been taken against smoking, it has been under pressure exerted initially by leaders of the medical profession. Thus it needs to be realised that the major responsibility for recommending and promoting action on tobacco products rests with medical bodies, as also emphasized in a paper by a leading member of this group.

Appropriate legislations by Central and State Governments are very powerful tools to influence tobacco behaviour as has been shown in several western countries. The paper on smoking control policy in U.K. by the Director, Action on

Smoking and Health mentions that regular increases in cigarette taxation has been the most effective single measure resulting in steady decline in consumption of cigarettes in U.K. without any fall in the government earning from tobacco. If anything, there has been an increase in the government's income with price rise of cigarette. In our own country, Cigarette Act which was enacted as early as 1975 requires a compulsory health warning that "Cigarette smoking is injurious to health" on every packet of cigarette and on every advertisement of cigarette. Unfortunately, however, no such mandatory warning is required to be displayed by bidi and chewing tobacco manufacturers on their products, which is a serious omission. Even with regard to health warnings on cigarette packs, we have made no innovative changes, unlike the west where for example, displaying levels of tar, nicotine and carbon monoxide on cigarette packets and advertisements, has been introduced. These issues are of a serious nature and need urgent attention.

In the long run, tobacco economics should receive serious attention and the possibilities of alternative uses of land and labour involved with tobacco cultivation and manufacture of tobacco products should be investigated. Action should be taken to ensure that where there is no commercial cultivation of tobacco, it is not permitted to develop and where it is a commercial crop, viable substitutes are found. The paper in the Appendix considers some of these issues in relation to bidi tobacco and makes a valuable contribution towards a better understanding of bidi tobacco economics.

In conclusion, high priority given to prevention of tobacco-related cancers in the National Cancer Control Programme needs to be translated in positive action by all concerned. No doubt infectious diseases are still rife in the country, but only if action is taken now, would the epidemic of tobacco-related diseases come gradually under control in the foreseeable future and the mounting accompanying costs would be reduced to a manageable proportion.

L.D. Sanghvi
Perin Notani

Section I

Burden of Tobacco Related Diseases and Deaths

Tobacco and Health : The Indian Scene. L.D. Sanghvi and Perin Notani (Eds.)
UICC Workshop, Tata Memorial Centre, Bombay 400 012, India. (1989)

Tobacco Related Cancers

L.D. Sanghvi[1]

Tobacco related cancers in India can be broadly classified on the basis of major risks of different tobacco habits. Tobacco chewing has a major risk for oral cancer; bidi smoking, for cancers of the pharynx and larynx and cigarette smoking, for lung cancer. Historically, tobacco chewing and hookah smoking were common habits in early part of the century. They have been gradually replaced by bidi smoking. Cigarette smoking which is a late comer on the Indian scene is also gaining momentum. On the basis of the data collected under the National Cancer Registry Project, it is estimated that almost one-third of all new cancer cases i.e. 190,000 cancer cases are related to tobacco habits. On the basis of these estimates, there will be at any given time about 500,000 cancer cases related to tobacco habits, leading to more than 100,000 deaths every year. Trends of different tobacco habits during the last two decades suggest that in the coming decades, cancer related to bidi and cigarette smoking will be increasing very rapidly.

Introduction

India ranks third in the world tobacco production. Of the 280 million kg. of tobacco cleared for home consumption per year (average for 1980–83), 86% was smoked, 13% was chewed and about 1% was used as powdered snuff. Among the smoking habits, bidi takes the leading position followed by cigarette. Other smoking habits include hookah (water pipe); chutta (a kind of cigar) and chilum (clay pipe) which are on their way out. Among the chewing habits, use of tobacco with pan (betel quid) or with lime are the common ones.

Several epidemiological studies carried out in the country have confirmed the high risk of tobacco habits for cancers of the upper alimentary and respiratory tracts. These cancers account for almost a third of all cancers in India. Risks of indigenous tobacco habits have also been identified for some other cancers (such as stomach and uterine cervix) which are in need of confirmation. Studies on chemical analysis of chewed tobacco, bidi and local cigarette have been carried out. They have revealed high levels of carcinogenic and toxic chemicals supplementing the epidemiological evidence. There is a changing pattern of tobacco habits in the country. Tobacco chewing and hookah smoking which were common habits prior to Second World War are being replaced by bidi and cigarette

[1] National Cancer Registry , ICMR, Tata Memorial Centre, Parel, Bombay 400 012, India.

smoking. These changes will have major impact on cancer incidence and pattern in the coming decades. All these aspects will be briefly reviewed in this paper.

Epidemiology

India has a long tradition of research in the field of cancer epidemiology. Earliest observations were made on the role of tobacco chewing habit in the aetiology of oral cancer which was very common in south India (1). Subsequent studies have confirmed its role not only in oral cancer but also in the cancers of the pharynx, larynx and oesophagus. Bidi smoking habit was already common during the Second World War. Its role in the aetiology of cancer was brought out in a case-control study carried out at the Tata Memorial Hospital in the early fifties (2). The study showed that bidi smoking had a significant role in the cancers of the oral cavity, pharynx and oesophagus like tobacco chewing habit, but its major effect was on cancers of the oropharynx. The study also showed that highest relative risks for all these sites were found in those persons who smoked bidis as well as chewed tobacco. Independent studies carried out in the country have confirmed these observations (3).

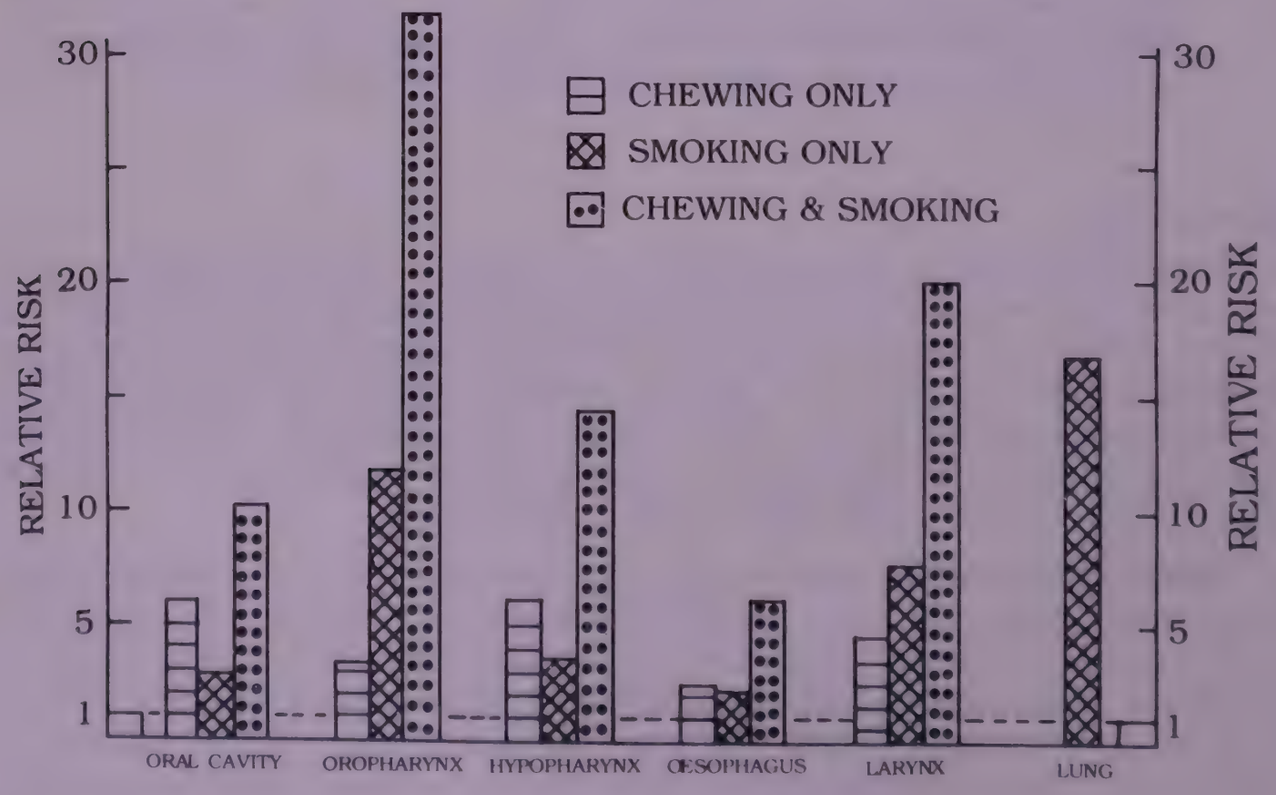

Fig. 1 Relative risk of developing cancers of the upper alimentary and respiratory tracts among persons addicted to chewing and smoking habits

Cigarette smoking habit has been a late comer on the Indian scene. Lung cancer became a visible problem in the Indian hospitals in the sixties and seventies. A study carried out in early seventies showed that risk of bidi smokers was equally high if not higher as cigarette smokers for developing cancer of the lung, with a dose-response relationship (4). This was confirmed in a subsequent study (5).

Latest available values of relative risks of different tobacco habits for cancers of the upper alimentary and respiratory tracts are shown in Figure 1.

Toxic chemicals in tobacco products

Evidence given above on epidemiology of tobacco related cancers has been supplemented by experimental and laboratory work on tobacco products. A brief summary of this work is given below (6–8) :

Chewing tobacco : Work carried on the two species of tobacco viz. *N.tabacum* and *N.rustica* has provided definite evidence of the presence of several N.nitroso compounds which are potent carcinogens. *N.rustica*, a tobacco species which is used mainly in chewing and smoking of hookah, has higher levels of tobacco specific nitrosamines compared to *N.tabacum*.

Bidi : In view of the high risk for cancers of the upper alimentary and respiratory tracts, chemical analysis of bidi smoke was carried out with the help of a U.S. laboratory in 1974. The results were very striking and showed that bidi delivered large amount of tar, nicotine and other toxic chemicals. Subsequent studies carried out in India confirmed these findings. It showed that a regular bidi (60 mm) delivers about 25 mg of tar and 2 mg of nicotine. A long bidi (75–80 mm) delivers 40 mg of tar and 2.5 mg of nicotine. Carbon monoxide level in bidi smoke is much higher than in cigarette smoke as tendu leaf used as a wrapper for bidi is much less porous than cigarette paper.

Cigarette : Indian cigarettes which have also been studied by us have revealed high levels of tar (19–28 mg) and nicotine (1.0–1.8 mg) compared to current levels in the West. The actual delivery of these toxic chemicals in the lung is much higher in India when it is taken into account that an Indian smoker takes 2 puffs per minute compared to 1 puff in the West. Filters used in Indian cigarettes are about half in length of those used in the West and their filtration efficiency is very low.

Burden of tobacco related cancer

National Cancer Registry Project of the Indian Council of Medical Research has provided estimates of the burden of tobacco related cancer in India (9). These estimates, which are given in Table 1, are based on the material for 1982 and 1983 from the three population-based registries in Bangalore, Bombay and Madras, and three hospital registries in Chandigarh, Dibrugarh and Trivandrum.

Table 1. Burden of tobacco related cancer in India, estimates for 1986.

	Males	Females	Total
Total population (millions)	390	370	760
Crude rate / 100,000	70	80	75
No. of new cancer cases per year	273,000	296,000	569,000
New cases related to tobacco habits :			
Mouth	30,888	24,642	55,530
Pharynx & larynx	42,159	9,435	51,594
Oesophagus	20,280	14,874	35,154
Lung	23,673	4,477	28,150
Others	13,000	6,572	19,572
Total	130,000 (48%)	60,000 (20%)	190,000 (33%)

Estimates are shown separately for males and females for commonly affected sites namely, mouth, pharynx and larynx, oesophagus and lung. Some other sites related to smoking habits known from the experience of the West such as bladder, pancreas etc. are taken as others. Stomach cancer for which one study has provided strong evidence of high risk of bidi smoking is not taken into account. Cancer of the uterine cervix for which cigarette smoking has been shown to have definite risk is also not included. It is likely that tobacco chewing and bidi smoking may show risks for cancers not studied so far. From these considerations, the present estimate of tobacco related cancers is a conservative one.

It is interesting to make a comparison of the burden of tobacco related cancers in India and in U.S.A. (10). In U.S.A. cancer mortality attributed to tobacco is 43% in men and 15% in women; overall mortality in the two sexes is 30%. In India, cancer morbidity related to tobacco is 48% in men and 20% in women with an overall estimate of 33% for the two sexes. Thus there is a general comparability of the burden of tobacco related cancers in the two countries. There is, however, a major difference in the cancer sites affected in the two countries. Whereas in U.S.A., 68% of tobacco caused cancer deaths were due to lung cancer, in India, 75% cancers related to tobacco were found in mouth, pharynx and larynx and oesophagus; lung cancer accounted for only 15% of cases.

Trends in tobacco consumption

Directorate of Tobacco Development, Ministry of Agriculture, Govt. of India provides annual figures of tobacco released for different purposes for home

consumption (11). Figures available since 1950 have been used here to calculate tobacco consumption per adult (aged 15 years and above) per year and are summarised in Figure 2.

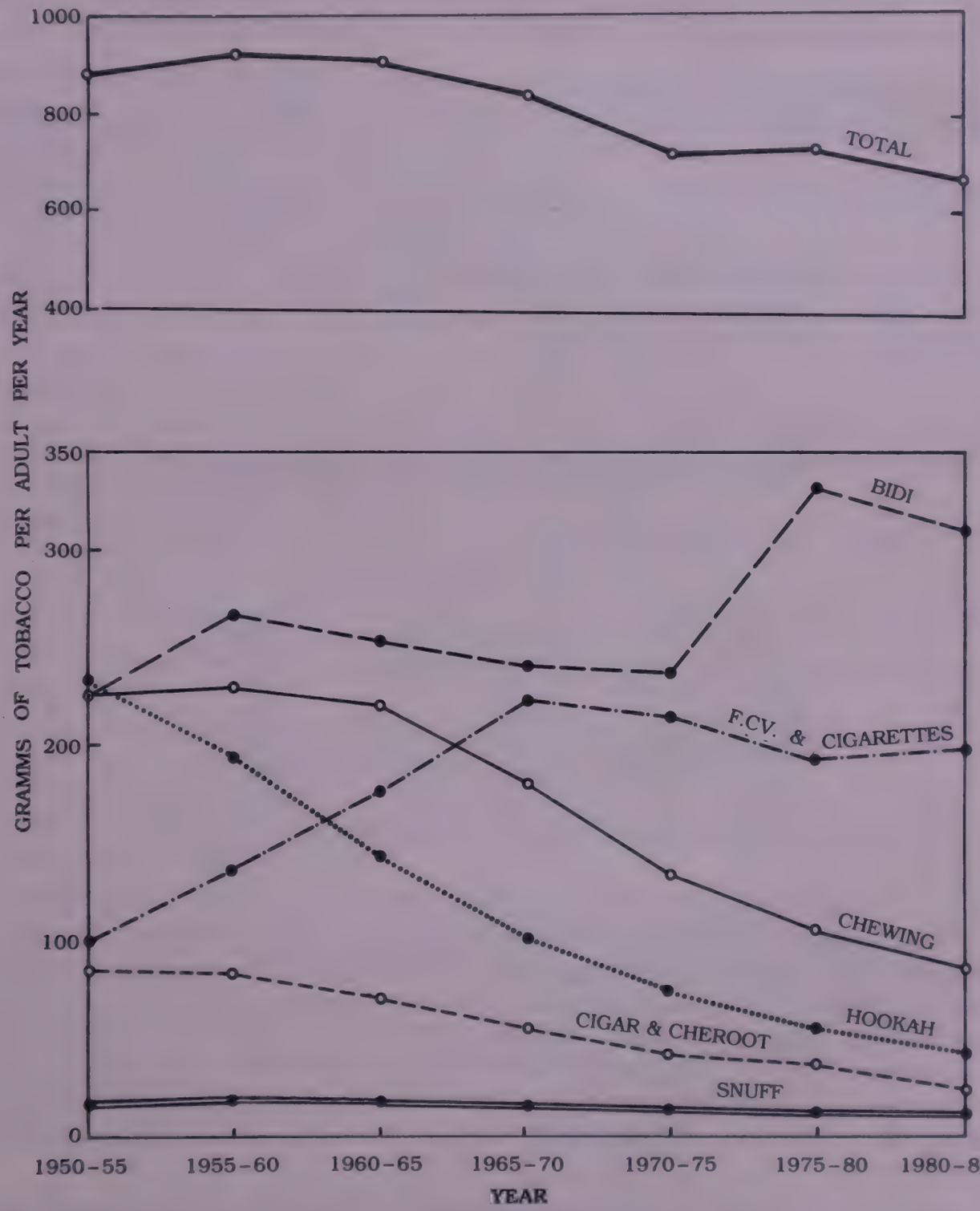

Fig. 2 Tobacco consumption in grammes per adult per year in India from 1950-1955 to 1980-1983.

It will be seen from the figure that during the period of three decades, tobacco consumption has declined from an average of 900 g during 1950–55 to about 700 g during 1980–83. There is also a major change in the consumption pattern during this period. Consumption for bidi and cigarette has gone up whereas consumption for chewing, hookah and for other purposes has declined.

Very little reliable information is available about the tobacco habit patterns in the population in recent years. On the basis of some careful surveys carried out in the past (12), the figures of tobacco consumption given above can be used to compute habit patterns in the population. Table 2 gives this type of reconstruction based on the following assumptions; bidi and cigarette smokers, smoke on an average 10 pieces per day, and chewers consume 3 g of tobacco per day. A cigarette on an average has 1 g of tobacco whereas bidi contains only 0.25 g Thus a cigarette smoker smokes 3.65 kg., a bidi smoker, 0.91 kg. and a chewer consumes 1.1 kg. of tobacco per year.

Table 2. Estimated number of adults (in millions) aged 15 years and over in India with different tobacco habits.
from 1950–55 to 1980–83

Tobacco habit	1950–55	1965–70	1980–83
Cigarette smoker	6	18	22
Bidi smoker	58	79	136
Tobacco chewer	47	49	31
Total population	230	300	400

Table 2 gives the estimated number of adults (in millions) aged 15 years and above with bidi, cigarette and tobacco chewing habits. Other tobacco habits which have declined in recent years have not been taken into account. No attempt has been made to separate the habits of males and females, although it is known that they are very different. Most of the bidi and cigarette smokers are males whereas almost an equal number of males and females chew a large proportion of tobacco. Among the males, 10–15% have both the habits i.e. they smoke bidis as well as chew tobacco.

There is a lag period of about three decades before the prevalence of tobacco habits gets translated as effect on the occurrence of cancer. Current estimates of tobacco related cancers given in the previous section have to be seen in the light of tobacco habits prevalent during 1950–55. Changing trends of tobacco habits during the subsequent periods suggest that with a decline of chewing habit, cancers of the oral cavity are likely to decline. On the other hand, cancers related

to bidi and cigarette smoking namely, pharynx, larynx and lung will be increasing very rapidly. These cancers are more difficult to detect and to cure than oral cancer.

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Smoking and Cardiovascular Disease

(With special reference to Cigarette vs Bidi Smoking)

S.P. Gupta[1]

Cigarette smoking has come to be universally recognized as a formidable factor in the morbidity and mortality of coronary heart disease (CHD). The few epidemiological studies available from this country reveal that both its prevalence and incidence may not be too different from that in the Western countries, at any rate, in large cities.

The pattern of smoking in India is quite different from that prevalent in the West, but precise data is meagre on the type and extent of smoking in different age-groups, in men and women and in various communities, urban and rural. Further, only preliminary information is available regarding differences in biological effects of cigarette and bidi tobacco. Our studies so far indicate that cigarette and bidi smoke have similar effect on platelet aggregation and blood sugar. Alterations of a somewhat lesser magnitude have been observed in both free fatty acid and left ventricular performance following smoking of bidis as compared to cigarettes.

Obviously there are wide gaps in our knowledge, and more work is needed to resolve the various issues in the association of indigenous tobacco habits and CHD.

Introduction

Smoking is universally recognized as one of the worst health hazards. In the successive annual reports of the U.S. Surgeon General since 1964, cigarette smoking has been identified as the single most important source of preventable morbidity and premature mortality. It has been estimated that an average of $5\frac{1}{2}$ minutes of life is lost for each cigarette smoked in cigarette smokers of 5-8 years duration (1).

Smoking has been linked with a number of diseases. During the first half of this century hazards of smoking chiefly centered round respiratory disorders viz., chronic obstructive airway disease (chronic bronchitis, emphysema), and lung cancer. However, while this association continues to hold true, the focus of attention during the past three decades has shifted to noxious effects of smoking

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Abbreviations : CHD : Coronary heart disease, COHb : Carboxyhaemoglobin, FFA : Free fatty acids, PEP : Pre-ejection period, LVET : Left ventricular ejection time.

on cardiovascular system, especially coronary heart disease (CHD).

Cigarette smoking and coronary heart disease–How strong is the association?

Cardiovascular diseases are currently regarded as the number one killer of mankind, and it is estimated that in the U.S. 30 to 40% of all deaths from CHD are attributable to cigarette smoking (2). The 1983 Surgeon General's report (2) states that "cigarette smoking should be considered the most important of the known modifiable risk factors for CHD in the United States" The pooled data from five large studies showed that men 40-59 years of age who were smoking a pack or more of cigarettes per day had a risk for a first major coronary event that was 2.5 times as high as that for non-smokers, with a strong dose-response relationship (3).

According to these reports, cigarette smoking confers approximately the same average increase in the risk of CHD as do hypertension and hypercholesterolaemia, but additionally, acts synergistically with these other risk factors on mortality from the disease. Further, women whose smoking patterns are similar to those of men have a similar increased risk of mortality and morbidity from CHD as compared with non-smokers. Following cessation of smoking, the risk of CHD attributable to smoking decreases by about 50% after one year, though it approaches that of a person who has never smoked only after a decade or more (2).

What is the data available from India?

Compared to the wealth of information on smoking and cardiovascular disease available from Western countries, very little is known about the prevalence or incidence of CHD in our population at large, and even less about the extent of smoking in our communities and its relationship with CHD. Gupta and Malhotra (4) reported the disease to be more than twice as common in urban area as in rural population in both men (45.3 and 17.2/1000) and women (28.1 and 12.6/1000). Table 1 gives the prevalence of CHD as reported in some of the Indian (4,5) and Western (6–8) studies and Table 2 shows the incidence of acute myocardial infarction based on a total population study in Rohtak city reported by us (9) (5.7/1000 for men and 2.3/1000 for women). This is an approximate estimate

Table 1. Prevalence of coronary heart disease

Prevalence rate per thousand in	Rohtak, India (Gupta) 1975	Chandigarh, India (Servothem) 1968	Framingham, U.S.A. (Kannel) 1976	Tecumseh, U.S.A. (Epstein) 1965	Busselton, Australia (Welbourn) 1969
Men	45.3	65.4	96.0	66.0	68.9
Women	28.1	47.8	38.0	47.0	58.0

Table 2. Incidence of acute myocardial infarction (Rohtak, Haryana)

	Men		Women	
Age group years	Population	Incidence per 1000	Population	Incidence per 1000
30–34	3722	0	3347	0
35–39	3371	0.8	3126	0.6
40–44	3646	1.3	2952	0.6
45–49	2338	6.4	1944	3.0
50–54	2137	9.3	1995	3.5
55–59	1714	10.5	833	2.4
60–64	1362	13.2	857	3.5
65–69	1033	13.6	750	4.0
70 & above	1342	18.6	1220	12.3
Total	20,665	5.7	17,024	2.3

of the disease incidence in small cities. The incidence in larger cosmopolitan cities would be higher,as in Chandigarh series (5), but even at this rate about 10,000 fresh cases of myocardial infarction can be expected annually (between 40-69 years of age) in the city of Delhi.

Pattern of smoking in India

Tobacco smoking amongst men is perhaps as widely prevalent in India as in Western countries, but the pattern of smoking is quite different. Tobacco is smoked by nearly all of the rural population as also the lower socio-economic groups in urban areas in the form of bidis, and by the middle and upper urban classes as cigarettes. Less common types of smoking include 'hukkah', 'chilum' and 'chutta' amongst the rural population, and cigar and pipe by the urban elite. Precise figures on the relative frequency of each of these types of smoking are available for only certain pockets of our population.

Relative risk of cigarette and bidi smoking

An average Indian bidi has about 330 mg of tobacco compared to 1 g in each cigarette. The effects of tobacco smoking on cardiovascular system are mediated through nicotine and carbon monoxide. Cigarettes and bidis marketed in India have a high nicotine yield (1.0–1.8 mg per cigarette and 1.7–2.9 mg per bidi) (10–11). Nicotine content of samples of tobacco used in our studies has been reported by Central Tobacco Research Institute, Rajahmundry to be higher in bidi tobacco compared to cigarette tobacco. Nicotine operates through increased levels of catecholamines which elevate heart rate and systolic pressure (thus increasing myocardial oxygen consumption), enhance platelet aggregation, and increase circulating levels of free fatty acids and blood sugar. We studied the effect of

smoking equivalent amount of cigarette and bidi tobacco and lettuce leaves (1 g each) wrapped in identical paper in a single blind manner on these parameters known to be involved in the pathogenesis of CHD. The results show that :

(a) Platelet aggregation and blood sugar increased significantly (especially the former) after both cigarette and bidi smoking, but there was no difference between two types of smoke.

(b) Free fatty acids (FFA) also increased after both cigarette and bidi smoking. The increase was slightly more in cigarette smokers than bidi smokers and both were much higher than in control (lettuce) smokers.

(c) No appreciable increase occured in serum cholesterol after either cigarette or bidi smoking.

One of the factors which would adversely affect platelet aggregation but not FFA is carbon monoxide in the tobacco smoke. Our preliminary work suggests that higher levels of carboxyhaemoglobin (COHb) result following smoking of bidi tobacco than an equal amount of cigarette tobacco, and this may account for the observed lack of difference in platelet aggregation, following cigarette and bidi smoking. However, Jindal et al (12) have reported higher levels of COHb in cigarette smokers. Obviously, more work needs to be done in this direction.

Precise data regarding the relative risk of cigarette and bidi smoking on CHD is not available from this country. In our urban–rural total population studies already referred to, no appreciable difference was seen in prevalence of CHD amongst smokers and non-smokers either in rural or urban group. A more recent case-control study from Bombay (13) has estimated a slightly greater risk for bidi smokers than cigarette smokers to develop CHD (bidi smokers : 3-fold, cigarette smokers : 2-fold) and myocardial infarction (bidi smokers : 4-fold, cigarette smokers : 3-fold) compared to non-smokers.

Acute effects of cigarette and bidi smoking on myocardial performance

How do these (and possibly many other) effects of smoking alter left ventricular functions. This was studied by us (14) on the basis of systolic time intervals (STI) in acute experiments, in both coronary and non-coronary subjects. The results show that :

(a) PEP/LVET ratio decreased ($P < 0.001$) in normal subjects (i.e., left ventricular performance improved), but lengthened in coronary patients ($P < 0.001$) after both types of smoke.

(b) The alteration in PEP/LVET ratio (and therefore, in myocardial functions) was less with bidi smoking in both groups of patients.

Effect of wrapping material

The wrapping material used in cigarettes and bidis is entirely different. Does this affect the quality of mainstream smoke which is inhaled? This was studied

(15) by comparing the effects of smoking 1 g bidi tobacco divided and wrapped in three cigarette papers with that of smoking three marketed bidis. No difference was observed in either FFA levels or platelet aggregation. COHb levels were appreciably higher after smoking marketed bidis, but the difference fell short of statistical significance. Thus the nature of the wrapping material does not appear to influence the quality of tobacco smoke.

Conclusions

Cigarette smoking has come to be universally recognized as a formidable factor in the morbidity and mortality of CHD. The few epidemiological studies available from this country reveal that both its prevalence and incidence may not be much different from that in the Western countries, at any rate, in large cities.

The pattern of smoking in India is quite different from that prevalent in the West, but precise data is meagre on the type and extent of smoking in different age-groups, in men and women, and in various communities, urban and rural. Further, only preliminary information is available regarding differences in biological effects of cigarette and bidi tobacco. Our studies so far indicate that while cigarette and bidi smoke resemble in respect of their effect on platelet aggregation and blood sugar, there are differences in some other parameters. Alterations of a somewhat lesser magnitude have been observed in both FFA and left ventricular performance following smoking of bidis as compared to cigarettes. More work is needed to resolve these issues.

Obviously, there are wide gaps in our knowledge, and research is required to obtain precise data on the various aspects of smoking mentioned above and its relationship with CHD and other cardiovascular diseases in this country. Keeping in view the urgency and complexities of the whole problem, a programme of Health Operational Research should be mounted starting from formulation of project(s), through collection and analysis of relevant data by a suitable sample, to testing and implementation of the solution. Ideally, such a programme should also include measures to help tobacco addicts quit smoking by conventional as well as non-conventional measures such as acupuncture, jogging (and possibly yoga), which can increase plasma beta-endorphin levels, since smoking addiction may be due to increased endorphin release.

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Studies on Coronary Heart Disease in Relation to Smoking

Perin N. Notani[1]

Cigarette smoking as a major risk factor for coronary heart disease (CHD) has been well documented in several studies undertaken in the western countries. In India, the predominant form of smoking is bidis. Bidi smoke has been demonstrated to deliver higher levels of tar, nicotine and carbon monoxide than western cigarettes. In view of these observations, it becomes very pertinent to study the health hazards due to bidi smoking. A cohort study undertaken in two socioeconomic groups showed that bidi smokers belonging to the blue collar group carried more than 3-fold risk of developing CHD and in particular myocardial infarct, as compared to the group without any tobacco habit. Similar, but slightly lower, risks for cigarette smokers were observed in this group.

Besides the epidemiological observations, there is also experimental evidence, reported in literature, to show that bidi smoke has equally deleterious effect as cigarette smoke on some of the parameters (pulse rate, blood pressure, platelet aggregation time and serum free-fatty acid levels), known to be related to the pathogenesis of this disease.

In addition to smoking, hypertension and elevated serum cholesterol levels are established risk factors. But, in view of the reported low cholesterol levels in the Indian population, smoking assumes the status of being the most important modifiable risk factor, which if eliminated from the population can result in prevention of almost 30%–50% of the new cases of coronary heart disease from occurring (in males in the age group 40 to 59 years).

Introduction

Several studies carried out in the western countries have implicated cigarette smoking as a risk factor for cardiovascular diseases, cancer of certain sites, in particular lung, and chronic obstructive lung disease. The first major prospective study was reported in 1958 by Hammond and Horn for the US population (1). In that study, overall, cigarette smokers were found to carry 70% greater risk of dying from coronary heart disease (CHD) than nonsmokers. The death rate was more than twice as great in men who smoked over a pack a day and the authors also

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Abbreviations : CHD : Coronary heart disease, MI : Myocardial infarct
COHb : Carboxyhaemoglobin, AIIMS : All India Institute of Medial Science.

noted a consistent dose-response relationship.

In the intervening 30 odd years numerous additional epidemiological studies, in geographically diverse regions, and involving different methodologies have been conducted, altogether representing 20 million person years of observation and they have come up with remarkably uniform results, that cigarette smokers have much higher death rate from CHD than nonsmokers (2).

In India varied forms of smoking besides cigarette smoking is prevalent. Bidi, obtained by wrapping 0·2 to 0·3 g of tobacco in tendu leaf *(Diospyros melanoxylon)* is the commonest and the most popular form. In fact, 85% of world's bidi production is accounted for by India. Furthermore, it has been shown that bidi smoke contains higher levels of the noxious components viz. tar, nicotine and carbon monoxide as compared to cigarettes (3, 4). Nicotine and carbon monoxide particularly contribute to the increased risk of cardiovascular disease in smokers.

In view of these observations, it becomes pertinent to review available information on the association of smoking, and bidi smoking in particular, with reference to CHD.

Epidemiological studies

Descriptive studies : Very limited information is available on the prevalence of the disease in the country. A survey in Haryana region carried out in 1970's covering the population in the age-group 30 and above, reported the prevalence to be much lower in the rural areas than semi-urban and urban areas,both in males (17.2, 45.3 and 65.4/1,000 respectively) and females (12.6, 28.1 and 47.8/1,000 respectively) (5, 6). These rates are comparable to those reported in earlier studies cited by Sapru (7).

Besides, a few studies describing the characteristic of CHD patients in a hospital set up have also been reported (8). In a study (9) of 100 young myocardial infarction patients from South India, smoking was considered to be the commonest risk factor, reported in 76% of patients. Hypertension, diabetes and cerebrovascular accident was ranked second (26%), obesity third (25%), and high serum cholesterol levels (more than 250 mg%) ranked fourth, being observed in 23% of patients.

Analytical studies :A case-control study carried out in patients attending the cardiology clinic at AIIMS, New Delhi, implicated cigarette smoking to be a risk factor for the CHD patients (10).

Concerted effort to study the role of bidi smoking was made in a case-control study (11), where the data on 185 CHD patients and matched controls were obtained from a hospital in Bombay. In this study, bidi smokers had a 3-fold risk for CHD and 3.8-fold for the subset of myocardial infarct (MI), compared to nonuser of tobacco, whereas cigarette smokers had 2.5-fold and 3.3-fold risk for CHD and MI respectively.

The only cohort study,that reports risks of bidi and cigarette smokers for

developing coronary heart disease, was carried out by us in two socio-economic groups. The methodology is described in detail elsewhere (12). Briefly, one cohort was of white collar workers, comprising of clerical and supervisory staff, junior and middle level executives, belonging to 19 organizations. The other was a blue collar group consisting of millworkers, mechanics, bus-drivers and conductors. Over six and a half thousand (6742) individuals of the white collar group and 5981 blue collar workers were enrolled in the study. As the study population belonged to organized groups, followup did not pose a serious problem. The period of followup varied from one to five years. Individuals in the cohort were kept under surveillance by regular six-monthly visits to the organizations. There was a loss to followup of 2% in the white collar group and 10% in the blue collar group.

Table 1. Estimates of rate ratios (RR) for incidence of coronary heart disease (CHD) and the subset of myocardial infarct (MI), for two cohorts (smokers vs. nonusers of tobacco).

Cohort▽ of	Type of smoker	Person years	Rate ratios with 95% confidence intervals			
			CHD		MI	
			RR	(n)	RR	(n)
Blue collar workers	Bidi	1512.5	3.27* (1.5, 7.2)	(18)	3.04* (1.1, 8.7)	(10)
	Cigarette	1128.5	3.01* (1.3, 7.1)	(12)	2.76+ (0.9, 8.3)	(7)
	Nonuser of tobacco	2211.0	1.00	(8)	1.00	(5)
White collar workers	Bidi	317.5	Sample size small			
	Cigarette	3046.0	1.13 (0.7, 1.8)	(26)	1.63 (0.9, 3.1)	(13)
	Nonuser of tobacco	10646.5	1.00	(84)	1.00	(29)

▽ Cohorts : All males 40–58 years, and attained age 41–59 years
* $P < 0.05$, + $P < 0.10$, (n): Actual number of cases observed.

Table 1 gives the estimated rate ratios (RR) for bidi and cigarette smokers to develop the disease for the two cohorts. The nonuser of tobacco did not smoke either bidis or cigarettes nor did he chew tobacco. The group of chewers and those with mixed habit is not considered here, since we were interested in studying the risk of smokers with single current usuage.

Looking at the cohort of blue collar workers (table 1), it is seen that bidi smokers had a 3-fold risk for developing CHD as well as for the subset of MI, whereas cigarette smokers had slightly lower risk of 2.8 for MI and 3.0 for CHD. These results are comparable to the results of the case-control study mentioned earlier (11), which was undertaken in a similar socioeconomic group.

In the cohort of white collar workers there were very few bidi smokers and the risk could not be assessed. The cigarette smokers had 63% greater risk of developing MI compared to the nonuser of tobacco, although no increase in risk was observed for CHD. However, it is seen from table 2, where the white collar and blue collar groups are compared,that there is no significant difference in risk of developing the disease in cigarette smokers of the two cohorts. The difference lies in the group that does not use tobacco, particularly for CHD. The nonexposed white collar worker has more than 2-fold risk of developing CHD compared to his counterpart in the blue collar group, suggesting that there is some factor in the life-style of the nonsmoker-nonchewer white collar worker which places him at a higher risk, for the disease. When, however heavy cigarette smokers ($\geqslant$ 20 per day) of white collar group are considered, a significantly higher risk for CHD (RR = 2.03, $P < 0.05$) as well as for the subset of MI (RR = 2.9, $P < 0.05$) is observed, compared to nonusers of tobacco.

Table 2. Estimates of rate ratios for incidence of CHD and of subset MI (white collar workers vs. blue collar workers)

White collar vs. Blue collar	Rate ratios with 95% confidence intervals	
	CHD	MI
Nonuser of tobacco	2.13* (1.03, 4.35)	1.19 (0.45, 3.13)
Cigarette smoker	0.76 (0.38, 1.54)	0.65 (0.25, 1.72)

* $P < 0.05$

Physiological effects of bidi and cigarette smoke

Besides the epidemiological observation that bidi smoker carries as high a risk as cigarette smoker for developing CHD, there is experimental evidence to show that bidi and cigarette smoke have equally deleterious effect on some of the parameters which are known to be related to the pathogenesis of the disease.

Gupta and Garg (13) have studied platelet aggregation time in seconds, serum free fatty acid levels and blood sugar levels in healthy smokers under strict experimental conditions (table 3). It is seen from table 3 that the effect on platelet aggregation time and blood sugar levels, 15 or 30 minutes after bidi and cigarette

smoke, are virtually identical. The increase in serum free fatty acid levels is also similar and is seen to be, 21.6%, 20.2% and 4.9%, 30 minutes after cigarette, bidi and control smoke respectively. The results were reported to be no different for bidi tobacco wrapped in cigarette wrapper or in the original marketed form (14).

Table 3. Mean, platelet aggregation time, serum free fatty acid levels and blood sugar levels in healthy bidi and cigarette smokers and controls[+]

	Mean ± SE Platelet aggregation time in seconds			Mean ± SE Serum free fatty acid levels in μEq/L			Mean ± SE Blood sugar levels in mg%		
	Control	Cigarette	Bidi	Control	Cigarette	Bidi	Control	Cigarette	Bidi
Before smoke	24 ± 6	24 ± 4	24 ± 3	532 ± 185	490 ± 135	486 ± 167	86 ± 15	84 ± 10	84 ± 11
15 Minutes after smoke	23 ± 6	17 ± 5	17 ± 5	551 ± 191	604 ± 196	576 ± 193	87 ± 14	94 ± 12	95 ± 12
30 Minutes after smoke	23 ± 7	24 ± 6	23 ± 6	558 ± 196	596 ± 215	584 ± 185			

Source : Gupta and Garg (1977).

[+]Equal amounts of bidi tobacco, cigarette tobacco and powdered lettuce leaves (control) were wrapped in cigarette wrappers and smoked.

The carbon monoxide (CO) inhalation during the act of tobacco smoking is the most obvious and dangerous exposure. Carbon monoxide has been reported to be as high as 7.7 vol% in bidis versus 3.5 vol% in western cigarettes (3). Furthermore, comparative studies of blood carboxyhaemoglobin (COHb) levels in bidi and cigarette smokers have revealed that COHb is very high in both types of smokers compared to nonsmokes. Jindal et al (15) have reported mean COHb levels in 81 cigarette smokers to be 9% and in 88 bidi smokers to be 7.4%, and both these levels were significantly higher than that measured in 58 nonsmokers which was only 0.8%. Earlier Bhown et al (16) had found the results of COHb levels of bidi smokers to be dose-related to the number of bidis smoked per day and in close conformity with those of cigarette smokers.

Bordia et al (17) have studied the effects of tobacco use on pulse rate, blood pressure, electrocardiogram and blood coaguability in healthy individuals and found the effect of bidi smoking to be as deleterious as cigarette smoking, for all the parameters studied.

Thus the epidemiological and experimental evidence are in the same direction, showing bidis to be atleast as harmful as cigarettes, if not more.

Other risk factors

Smoking along with hypertension and elevated serum cholesterol levels are

the three established risk factors for cardiovascular disease and smoking acts synergistically with these factors (2).

Several workers, however, have shown that serum cholesterol levels in healthy individuals in the Indian population are generally much lower than their counterparts in the western population, especially in the low income groups (18).

Table 4. Mean serum cholesterol levels (mg/100cc) in healthy individuals

Country	Author	Age groups (Years)			
		21–30	31–40	41–50	⩾ 51
India	Vengsarkar et al (1968)	152 ± 44		168 ± 36	
India	Padmavati et al (1961)	164 ± 25	174 ± 40	172 ± 23	150 ± 30
India	Srikantia et al (1961)	161 ± 10	173 ± 5	175 ± 5	174 ± 16
U.S.	Kannel et al (1964)		219 ± 45 222 ± 42	229 ± 46 231 ± 39	
Italy	Keys et al (1954)		228 ± 41	231 ± 40	

Source : Vengsarkar et al (1968).

Between the ages of 31 and 50, it is seen from Table 4 that the mean serum cholesterol levels vary between 172 mg/100 cc to 175 mg/100 cc in the Indian population groups whereas in the comparable age-groups in western populations it ranges between 219 mg/100 cc to 231 mg/100 cc. Infact Vengsarkar et al (18) noted that in their group of CHD patients the mean serum cholesterol levels were lower than the normal values reported for healthy individuals in the west. Dolder and Oliver (19) in their study of risk factors operating in young men with myocardial infarct, from nine countries, noted that high serum cholesterol and triglyceride levels were the common features of the cases from all 7 developed countries but was not so for cases from Bombay and Singapore, and the values were consistently on the lower side of the range.

Thus for the Indian population, smoking – both bidis and cigarettes – may be considered the most important of the known modifiable risk factor for this disease.

Assessment of attributable risk and conclusion

If smoking (bidi and cigarette), as mentioned above, emerges to be an important risk factor, it is of interest to assess, what proportion of the disease is avoidable, if smoking could be eliminated from the population. This proportion is

not only dependent on the excess risk of morbidity that smokers carry for the disease but also on the fraction of the population that smokes. It is given by the expression $[P(I_1 - I_0) / (PI_1 + (1-P)I_0)]$, where I_1 and I_0 are incidences of the disease in smokers and nonsmokers respectively and P is the fraction of the population that smokes. In absence of incidence data, the relative risk ($RR = I_1 / I_0$) can be substituted in the formulae to obtain the attributable proportion, known as attributable risk percent, as $[P(RR-1)/(1 + P(RR-1))] \times 100$.

Table 5. Attributable risk per cent for CHD in males

P	Attributable Risk	
	RR = 2	RR = 3
	%	%
0.40	29	44
0.50	33	50
0.60	38	55

P = fraction of smokers in the population
RR = Incidence rate ratio

The attributable risks obtained by taking combinations of different values of relative risk (RR), and of fraction P of smokers in the population, are shown in Table 5. It can be seen for example, that for a relative risk of 3 (as estimated in table 1 for male bidi and cigarette smoker in age group 41–59 years) and P the fraction of smokers in the population as 50% (observed for blue collar group) almost half the new cases of CHD in this group (males, 41–59 years), are attributable to smoking–if the excess is mainly due to smoking habit–and hence avoidable if smoking could be eliminated from the population.

The association with smoking is further strengthened by the fact that there is a substantial decrease in CHD mortality in those who quit the habit, as reported from studies in the west (2). Approximately 10 years following cessation of cigarette smoking, CHD death rates in those who used to smoke less than a pack a day was virtually identical to that of lifelong nonsmokers and for ex-smokers of more than a pack a day there was a reduction proportional to lifetime exposure.

It needs to be added, in conclusion that the benefits of elimination of smoking can not only be translated in reduction in risk of smokers for developing cardiovascular disease but a host of other conditions including cancers of certain sites and chronic obstructive lung disease.

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Tobacco Smoking and Non-neoplastic Respiratory Disease

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Chronic bronchitis and emphysema are the most common non-neoplastic respiratory diseases associated with all the major types of smoking in India (bidi, cigarette, chutta and others). This is the result of structural and functional changes both in the conducting airways and the pulmonary parenchyma produced by tobacco smoking.

Surveys have been carried out by PGI in Chandigarh city and a neighbouring rural area using a standardized questionnaire along with ventilatory function measurements. It was found that smoking was more prevalent in males than females and in rural than in urban area. Furthermore, the prevalence of chronic bronchitis was higher in rural male smokers (16.4%) than urban male smokers (9.9%). Similar studies in Shimla Hills and South-East Orissa revealed that smoking in adult females was also high. Infact 85% of females in the Orissa survey smoked chutta compared to 30% of males, and chronic bronchitis was equally prevalent (33%) in the smokers of both the sexes. Female smokers of rural Shimla exhibited the highest prevalence (52%) of the diseases.

Effect of smoking on other conditions, like tuberculosis of lung, and non-neoplastic respiratory problems of passive smokers are also discussed.

Introduction

There is a lot of clinical, epidemiological and experimental data to prove the relationship between cigarette smoking and a wide range of diseases (1). Not only smoking is directly responsible for the cancer of the lung and other sites, but also for increased morbidity and mortality due to non-neoplastic respiratory diseases like chronic bronchitis emphysema syndrome, cor pulmonale and respiratory infections. The evidence linking smoking and many diseases is so certain that the 1983 U.S. Surgeon General's Report on Smoking and Health concluded that cigarette smoking is the largest avoidable cause of death and disability in United States.

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Abbreviations : FEV_1 : Forced expiratory volume per second, COLD : Chronic obstructive lung disease.

Non-neoplastic effects of smoking on lungs are discussed below with special reference to India.

General

Smoking affects both the lung structure and function in several ways. Some of these are discussed below :

Structural changes : There is increased mucous formation due to hypertrophy and hyperplasia of mucous glands of the large airways. This results in increased sputum production. It is mainly the tar yield of cigarettes which affects the phlegm (2).

In the small airways, there is inflammation, goblet cell hyperplasia and intraluminal mucous formation which results in airways closure. This may be the forerunner of chronic obstructive lung disease.

The bronchial epithelial permeability is also abnormal in smokers as measured by radioisotope technique and there is rapid improvement in this abnormal permeability after cessation of smoking (3). It has been earlier demonstrated experimentally in guinea pigs that smoke damages the tight junction of airway epithelium (4). The increased permeability results in increased absorption of inhalent toxins and other aerosols. For example, bronchial responsiveness of inhaled bronchoconstrictors (e.g. methacholine) is markedly increased in both symptomatic and asymptomatic smokers (5, 6).

Smoking also causes destruction of alveolar walls resulting in centrilobular emphysema. This may be the result of increased influx of neutrophils, which release neutrophil elastase and also inactivate alpha-1 antiprotease. Both the factors promote proteolysis.

Functional changes : Small airway closure is manifest by increases in closing volume and other sensitive tests of small airways function after about 15 years of smoking even in asymptomatic subjects. The expiratory flow rates are impaired and FEV_1 can be used as a predictor of the development of obstructive airway disease. A relatively low FEV_1 by middle age and a faster rate in its fall with age, indicates a smoker who is likely to develop severe functional impairment.

The fall in FEV_1 can be significantly arrested by discontinuation of smoking. Even if smoking is stopped at the age of 40 years the rate of FEV_1 fall in ex-smokers becomes similar to that in non-smokers.

Chronic bronchitis and emphysema

It is now well established that chronic bronchitis and emphysema (often labelled collectively as chronic obstructive lung disease, COLD) are smoking related diseases. As the pattern of smoking in women approaches that in men, in the West, the incidence of the disease in the females rises and may some day equal the male incidence.

Evidence from the recent literature suggests that it is not possible to differentiate clinically between chronic bronchitis and emphysema (7,8). There were no differences in the degree of mucous gland hyperplasia on post mortem studies in the two conditons and severe emphysema was present in 50% of clinically diagnosed type B bronchitics and 70% of emphysema patients. The classical gas exchange disturbances of emphysema were also seen in chronic bronchitis (7). Similarly, the radiological differences were also overlapping. Since both of these nearly always coexist, these are better labelled at least clinically, as chronic bronchitis and emphysema (8).

Table 1. PGI studies on prevalence of chronic bronchitis in adults

Population group	Total No.		Smokers (%)		COLD (%)			
	M	F	M	F	Males		Females	
					Sm	Ns	Sm	Ns
* Bhiwani, rural (1975)	278	–	70.9	–	21.8	–	–	–
* North India, general (1974–78)	2360	686	33.0	–	20.0	5.2	–	4.1
* Chandigarh, teachers (1976)	397	346	15.4	–	8.2	0.6	–	1.2
* Chandigarh, city (1977)	1450	1375	30.6	–	9.9	0.9	–	1.6
* Chandigarh, rural (1980)	671	898	42.7	1.4	16.4	4.1	–	5.0
* Shimla, rural (1984)	304	142	60.5	28.2	32.0	5.8	52.0	6.0
* Orissa, (1983)	169	205	30.2+	84.9+	33.3	–	32.7	–

Sm : smokers; Ns : non-smokers + chutta smokers

Studies from India : Incidence of chronic bronchitis and emphysema on the basis of a clinical questionnaire has been reported to be high, especially in smokers (9–11). At Chandigarh, about 19% of our total chest clinic attendance

comprises of chronic obstructive lung disease, excluding asthma. Our earlier investigations, on healthy attendants of sick patients in the hospital and other volunteers on whom a standardized questionnaire (based on British Medical Research Council questionnaire) was filled and an objective measure of peak expiratory flow rate (PEFR) obtained, revealed the presence of chronic bronchitis in 5.2% of male non-smokers and in 20% of male smokers (12, 13). Subsequently, field studies were undertaken in a part of the city of Chandigarh and in a nearby village Mullanpur, utilising the same method (14). There were 30.6% male smokers in Chandigarh and 42.7% in the village Mullanpur. The prevalence of chronic bronchitis in male smokers was seen to be 9.9% at Chandigarh and 16.4% in the rural area. Corresponding figures for non-smokers were 0.9% and 4.1% respectively. In non-smoking females the prevalence of chronic bronchitis was 1.6% and 5.0% respectively in the city and rural area (14).

The frequency of major respiratory complaints (cough, sputum, shortness of breath, wheezing) and of established chronic bronchitis, was higher in the rural group especially in male smokers. Both groups (city and rural) showed a positive age related gradient in chronic bronchitis and this was more obvious in the rural adults.

Surveys conducted at different places reveal a prevalence of chronic bronchitis in male smokers to vary from 8.2% in Chandigarh teachers to 33.3% in chutta smokers of Orissa (14 – 16) as seen in Table 1. Smoking was common in females at two places viz., South East Orissa (chutta smoking) and rural Shimla (bidi smoking). In the female bidi smokers, the prevalence of chronic bronchitis was as high as 52.0% and in chutta smokers it was 32.7% (Table 1).

Exercise tolerance

Smoking reduces the exercise tolerance even in asymptomatic individuals. Even a single cigarette or bidi smoking has been shown to increase airway resistance (17). Sportsmen who smoke, significantly reduce their work capabilities in competitive sports.

Exercise tolerance of bronchitic patients is further reduced with continued smoking and this has been attributed to high carboxyhaemoglobin level (18). Our own studies have shown high carboxyhaemoglobin levels of 7 – 8% not only in cigarette smokers but also in bidi smokers (19).

Tuberculosis of lung

Association between tobacco smoking and tuberculosis has been described (20 – 21). Smoking may be an important cause of the breakdown of healed or quiescent respiratory tuberculosis lesions in adults and may account for considerable part of the excessive male mortality in middle and late life (22).

Miscellaneous effects

There is an increase in incidence of respiratory infections especially pneumonia

and influenza in smokers. Post operative respiratory complications are also more in smokers. Smoking has been considered to be an important cause of spontaneous pneumothorax.

Asthmatics get exaggerated symptoms when they smoke and asthma attacks may be induced. The outlook for asthma worsens in proportion to the number of cigarettes smoked (23).

Smoking has synergistic effect with certain occupational exposures, like coal mine dust, grain or cotton dust, in producing chronic bronchitis and airways obstruction. Therefore, smoking is a powerful risk factor which increases the risk not only of lung cancer but also airway obstruction associated with occupational exposure.

Non-neoplastic respiratory problems in passive smokers

Non-smokers, exposed to side stream smoke in the company of smokers, are referred to as passive or involuntary smokers. Children, non-smoking spouses and work colleagues of regular smokers belong to this category. It is now known that passive smoking is harmful and is an important risk factor for lung cancer and other respiratory problems.

In children, the rate of lower respiratory illnesses is linearly related to maternal smoking (24). There is twice the risk of pneumonia and bronchitis amongst children of smokers during first year of life (25). Heavy smoking during pregnancy may have a direct effect on the offspring's subsequent lung function and a relationship was suggested between early childhood bronchitis or pneumonia and impairment of lung function in later childhood with maternal smoking habit (26).

In adults, cough, breathlessness, chronic bronchitis and induced asthma attacks are more common in nonsmokers exposed passively to tobacco smoke as compared to those who are not exposed (27).

Our preliminary investigations point towards increased bronchial reactivity to inhaled bronchoconstrictor aerosols in wives of smokers presumably due to increased epithelial permeability. This would imply increased absorption of most tobacco combustion byproducts which these subjects inhale in the company of smokers. This in addition to other factors may explain at least partially prevalence of chronic respiratory disease and lung cancer in non-smoking subjects especially women in this country (28).

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Chronic Obstructive Lung Disease in India- Morbidity and Mortality due to Smoking*

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It has been established that cigarette smoking is the major cause of COLD in U.S. In India, bidi which is different from cigarettes in several ways, is the most prevalent form of smoking. Hence, there is a need to assess the proportion of COLD cases in the country that are attributable to smoking.

With the limited data available, morbidity due to COLD in the country for the 1986 population (760 million), has been estimated to be in the range of 15 to 29 million and mortality between 0.25 to 0.46 million. Furthermore, it has been shown that between 7 and 13 million prevalent COLD cases and 0.11 to 0.21 million COLD deaths are attributable to smoking.

Introduction

The U.S. Surgeon General's Report (1) examined the relationship of tobacco use in the development of COLD in 1984, and came to the conclusion that cigarette smoking is the major cause of COLD in U.S.

In India, as the most common form of smoking is the bidi, which is different from cigarettes in several ways, there is a need to evaluate the role of bidi smoking in the development of COLD and assess the proportion of cases in the country that are attributable to smoking.

Classification of lung diseases

Chronic obstructive lung disease-COLD literally means obstruction of airflow within the lungs. There are many conditions in which airways obstruction is common. Ciba guest symposium (2) subdivided these groups of diseases into two categories, the reversible obstructive lung disease like bronchial asthma, and the irreversible obstructive lung disease under which are included chronic bronchitis and emphysema. However, emphysema, which often coexists with bronchitis is defined on the basis of morbid anatomical condition. As clinically the two

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abbreviations : COLD : Chronic obstructive lung disease.

* The paper was not formally presented but is included for completeness of coverage of ill-effects of tobacco-use.

conditions cannot be differentiated, they are usually considered together. This classification is generally accepted and the Report of the U.S. Surgeon General (1) also includes chronic bronchitis and emphysema under chronic obstructive lung disease (COLD).

Furthermore, the Report states that COLD comprises of three separate disease processes.

a) "Chronic mucus hypersecretion leading to chronic cough and phlegm"

b) "Airway thickening and narrowing with expiratory airflow obstruction" and

c) "Emphysema which is characterised by abnormal dilation of the distal air spaces along with destruction of alveolar walls'.

The term mucus hypersecretion is vague. In order to have uniformity in diagnosis, the Medical Research Council of Great Britain has developed a standard questionnaire which has been extensively used all over the world including India. The other two disease processes mentioned above develop symptomatic ventilatory limitation which is quantifiable, and is estimated mainly by forced expiratory volume per second (FEV_1).

Morbidity

Morbidity due to COLD is a better indicator of the impact of disease on the society than mortality, as the progressive loss of lung function that characterises COLD can lead to severe shortage of breath, limiting the activity level (1).

Morbidity in developed countries

Several studies in the west have estimated the prevalence of COLD. In a nationwide sample of 11600 men and women in the United States (3) crude prevalence of chronic bronchitis was 3.1% in men and 3.4% in women. The corresponding estimates for emphysema were 1% and 0.3% respectively. Other studies in U.S. have reported crude prevalence varying from 8.6% to 23.5% in males and 1.1% to 8.1% in females (4–6).

Prevalence of chronic bronchitis in U.K. is reported to vary from 11.2% to 32.8% depending on the study area (7, 8). In the European countries, it varies from 9% to 28% (9, 10).

Morbidity in India

A number of surveys conducted in India have shown a wide variation in the prevalence of chronic bronchitis (0.8% to 8% in males and 0.6% to 4.3% in females) (11–13). The early studies adopted merely the standard British questionnaire for screening. However, in studies in specific groups of adults in Chandigarh (northwest India) where airway obstruction was also determined by objective tests, the crude prevalence of chronic bronchitis ranged from 3.7% to 10% in males and from 1.6% to 5.2% in females. In industrial groups prevalence was as high as 12.5% (14–17).

Urban/rural as well as regional variation have been studied. Malik from a study in one part of urban Chandigarh and an adjoining rural area concludes that prevalence of chronic bronchitis is higher in rural areas both in males (9.4% vs. 3.7%) and in females (5.2% vs. 1.6%) (17). In contrast, for Delhi (north India), a reverse situation is indicated. Urban prevalence (8.0% in males and 4.3% in females) is higher than prevalence in rural Delhi (4.7% in males and 3.5% in females) (17). Some workers, however, report that the disease is equally common in both the areas (18). The observed inconsistencies may well be due to comparison of prevalence rates which are not adjusted for age and other confounding variables like pollution. On the basis of available data, it is not possible to reach a definitive conclusion regarding urban-rural differences. On the other hand, it is generally believed that the disease is less common in the southern than in the northern parts of the country (18). In a study in Madras city (south India) the observed prevalence was only 1.4% in males and 2.7% in females (12). There is also indirect supportive evidence as seen from the high proportion of cor pulmonale in cardiac cases in the north compared to the south (18).

For estimating the magnitude of the problem in the country, Sapru (19) assumed an average prevalence of atleast 3%. By considering prevalence in all the southern states as that of Madras city and in all the northern states as that of Chandigarh, we obtained a rough estimate of 5% for the prevalence in the country. However, a better approach would be to estimate a plausible range for prevalence of COLD from the available data. It was found that the study of Malik and Wig (15) on healthy adults accompanying sick patients covers unselected persons in the area, is sufficiently large, is based on objective tests and gives age-sex-specific rates for smokers and nonsmokers. In view of the complete and reliable information given, this study has been chosen for the various estimates obtained in this report. However, in extrapolating the age-adjusted rates computed from this study, (viz., 11.2% in males and 4.8% in females) to the 1986 population of 760 million in the country, the variation (within region and between regions) needs to be taken account of. The proportion of population having rates similar to Chandigarh is not likely to exceed 50% (as it is roughly the proportion of population in north India).

It is more difficult to assign a lower bound – perhaps, the population having rates similar to Chandigarh, would not be less than 20%. The next step is to choose the prevalence for parts of the population, which do not have rates similar to Chandigarh. As we have already seen, wide variation is reported for prevalence of chronic bronchitis. It is pertinent to mention here that one study showed that the prevalence of chronic bronchitis was 5.5% as determined by the clinical questionnaire alone and 8% with simultaneous objective measurement of airflow obstruction (17). This indicates that the early reports especially those with very low rates are not reliable. It is unlikely that the variation in prevalence of COLD in the country is more than 4-fold. In other words prevalence in any part is not likely to be lower than 1/4 the prevalence in Chandigarh.

Applying the above-mentioned prevalence rates of 11.2% in males and 4.8% in females to a fraction P equal to 0.5, 0.3 and 0.2 of the population and half, one-

Table 1. Alternative estimates for prevalence and mortality due to COLD in the country[+]

Alternative		Prevalence (%)		Estimated number of cases (million)	Estimated number of deaths (million)
	(1 – P)[++]	Males	Females		
1	0.5	5.6	2.4	28.7	0.46
2	"	3.7	1.6	25.5	0.41
3	"	2.8	1.2	24.0	0.38
4	0.7	5.6	2.4	24.9	0.40
5	"	3.7	1.6	20.4	0.33
6	"	2.8	1.2	18.2	0.29
7	0.8	5.6	2.4	23.0	0.37
8	"	3.7	1.6	17.8	0.28
9	"	2.8	1.2	15.4	0.25

[+] Estimates are for the adult population (15 years and above) for the year 1986.

[++] Age-adjusted prevalence rates for Chandigarh, i.e. 11.2% in males and 4.8% in females, are applied to a proportion P of the population and to the remainder i.e. to a proportion (1 – P) of the population, the rates shown in the body of the table are applied to estimate various alternatives for prevalence in the country.

third and one-fourth the above rates to the remaining (1 – P) fraction i.e. 0.5, 0.7 and 0.8 respectively of the population, various estimates of prevalence of COLD in the country have been obtained and are shown in Table 1. It can be seen that these alternatives give a range of 15 to 29 million as the prevalence of COLD in the country corresponding to average prevalence varying from 3.2% to 6.1%. We feel that an average prevalence of about 5% is likely to be a reflection of the true prevalence in the country.

Mortality

As mentioned earlier, COLD is due to 3 disease processes,mucus hypersecretion, airway narrowing and emphysema. The extent of the damage due to these processes varies from individual to individual. However, ventilatory limitation causes much greater risk to life compared to chronic cough or phlegm.

COLD progresses slowly and death in an individual usually occurs after a prolonged symptomatic period. It is reported (1) "that many individuals with COLD die with their disease rather than because of it". Furthermore, it has also

been observed that inspite of COLD having played an important role in a person's death, it does not always get recorded as a cause of death either in hospital records or death certificates (20). Thus the recorded number of COLD deaths is likely to be an under-estimate of true deaths due to COLD.

Mortality in developed countries

The age-adjusted mortality rates for COLD in 1980 in United States were 26 per 100,000 in white males and 8 per 100,000 in white females (1).

Mortality in India

Death records for Bombay city provide the only reliable data on mortality due to COLD (21). However, deaths due to chronic bronchitis, emphysema and asthma are clubbed together in reporting cause of death. It is generally accepted that prognosis is good for asthma and mortality is very much lower than for chronic bronchitis or emphysema. Therefore, in the absence of more precise data, mortality rates given for the combined group of chronic bronchitis, emphysema and asthma will be considered as mortality due to COLD for the purposes of extrapolating the mortality rates for the country.

As the prevalence of COLD is supposed to show regional variation (as explained above), extrapolation of the age-specific mortality rates of Bombay, directly to the country would perhaps not be valid. The issue has been resolved by considering an acceptable estimate for the ratio of mortality to morbidity due to COLD (as actual data for computing this ratio is not available for any part of the country). Of the 3 such alternative ratios viz., 0.5, 1.1 and 1.6 percent obtained on the assumption of prevalence of COLD in the population of Bombay being similar, half and one-third the prevalence of Chandigarh, the first alternative (0.5%) seems to be unlikely as the corresponding ratio for U.S. is 0.6%. (1). The estimates for the number of deaths due to COLD in the country on the basis of the remaining two ratios (applied to the earlier estimated prevalence of 15–29 million) vary from 0.16–0.32 million for one ratio (1.1%) and 0.24–0.46 million for the other (1.6%). A critical study of the resulting death rates due to COLD in adults in the country based on the above estimates of mortality shows the mortality to morbidity ratio of 1.6% to be more acceptable.

Estimates for mortality are worked out on the basis of mortality to morbidity ratio being 1.6% and shown in table 1. Thus the mortality due to COLD in the country is estimated to be in the range of 0.25 to 0.46 million.

Sapru (19) has not estimated deaths due to COLD. However, he estimated deaths due to non-tuberculous chest diseases as 14.6% of total deaths based on the findings in a rural survey (22). In the same survey, the corresponding percentage in adults was 13.4%. If we assume that the proportion of COLD deaths in non-tuberculous chest diseases in adults in the country is similar to that observed in Bombay (44.5%) (21), the estimate for COLD deaths works out to 0.30 million and is covered by the estimated range given above.

Smoking practices in India and product evaluation with regard to relevant harmful constituents of smoke

In India various forms of smoking are practiced, bidi being the most popular form. Other indigenous forms like hukkah, chilum, cheroot are smoked mostly in rural areas. Cigarette is the preferred form of smoking in urban areas.

The overall toxicity as measured by total particulate matter (tar) and nicotine shows that bidis yield high values of tar (23 mg–41 mg per bidi) and nicotine (1.7 mg–2.8 mg per bidi) under standard conditions modified for bidi. The carbon monoxide in bidi smoke is 7.7 vol percent. Furthermore, hydrogen cyanide which is the strongest cilia toxic agent in tobacco smoke is also high in bidi smoke (688μg–904 μg per bidi) (23–25).

Popular brands of cigarettes marketed in India yield 19 to 28 mg of dry TPM, 0.94 mg to 1.78 mg of nicotine and 366μg to 638μg of hydrogen-cyanide per cigarette under standard international smoking conditions (23, 25). Filter tip cigarettes in India do not necessarily have lower values of tar and nicotine than nonfilter cigarettes (23). It is also worth noting that cigarettes currently marketed in India are akin to cigarettes marketed in the U.S.A. in the sixties.

A single chutta, when smoked under modified standard conditions for bidis, yields 44.8 mg of tar and 5.25 mg nicotine whereas a hookah when smoked for 35 minutes yields only 9.1 mg of tar and 0.55 mg of nicotine (25). Emission levels for Chilum (a clay pipe) are not available.

Smoking and COLD

Experimental and epidemiologic studies have shown beyond doubt that cigarette smoking is the major cause of COLD. In nonsmokers clinically significant COLD is rare (1).

The irritants in the smoke are found to cause airway resistance which progressively leads to pathologic changes in the small airways, as demonstrated by various tests. There is a significant decrease in airway conductance, maximum mid expiratory flow rate and forced expiratory volume per second in smokers (1).

In India several studies have been conducted to assess the effect of bidi smoking on physiologic parameters related to pathogenesis of COLD.

It is reported that in bidi smokers, the immediate effect of smoking is seen more on central airways whereas the effect on peripheral airways is equal in both bidi and cigarette smokers (26). From these observations it is concluded that, (a) risk of COLD in bidi and cigarette smokers is likely to be similar, (b) disease may manifest earlier in bidi smokers and (c) ventilatory defect may be higher in cigarette smokers (as shown earlier (27)).

Pathogenesis of lung damage

The association of cigarette smoking and mucus hypersecretion has been well demonstrated over the past 20 years (28). This is probably due to the inflam-

matory changes in large airways. Respiratory bronchiolitis, small airway inflammation, fibrosis and loss of surrounding alveolar attachments which lead to airflow limitation have been identified in smokers (29–31). Though the exact mechanism is not clear it is most probable that proteolytic damage from leukocyte and macrophage elastases may be responsible.

There are three major pathogenetic possibilities by which smoking may damage the lungs (28). They are : (1) altering protease-antiprotease balance in the lung, (2) compromising immune mechanism and (3) interfering with pulmonary clearance mechanisms.

Proteolytic lung damage : Cigarette smoke attracts alveolar macorphages to cluster around small terminal bronchioles, the aveolar macrophages release neutrophil–chemotactic factor which attracts polymorphonuclear leucocytes to this site from within the circulating blood stream. The polymorphonuclear leucocytes then release human neutrophil elastase, a proteolytic enzyme which destroys the elastic tissue of the lung.

Interference with immune mechanism : Inhalation of tobacco smoke produces significant changes in cellular and humoral immunity in both animal and man. However, the role of such changes in pathogenesis of lung disease remains speculative. Macrophages from smokers respond abnormally to migration inhibitory factor or antigen challenge. T lymphocytes obtained by bronchoalveolar lavage in smokers showed a diminished response to phytohemaglutinin thereby showing that cigarette smoking may impair cellular immune defenses.

Effect on clearance mechanism : The mucociliary transport system protects the lung against inhaled particulate matter. Most evidence indicates that long-term smoking reduces mucociliary transport, thereby making the lungs susceptible to infection and injury due to inhaled particles.

Risk associated with smoking : studies in developed countries

The risk of chronic bronchitis in smokers compared to nonsmokers varied from 4.1 to 5.3 in U.K. (7, 8) and from 2.9 to 5.4 in U.S.A. (32, 5).

The lung function which declines with age shows a steeper decline in smokers as a result of progressive lung damage which leads to symptoms of COLD and possible premature death. Consequently, the risk of death from COLD increases with early age at initiation, frequency of cigarettes smoked and inhalation. In the U.S. veterans study, mortality ratio of chronic bronchitis, emphysema, and chronic bronchitis alongwith emphysema were 3.6, 5.3 and 4.8 respectively in light smokers (1-9 cigarettes) and increased to 8.3, 25.3 and 22.0 for heavy smokers (40+) (33, 34). In the study on British physicians (35) mortality ratio for chronic bronchitis and/or emphysema was 17, in males smoking 1–14 cigarettes, 26 in those smoking 15–24, and 38 in those smoking 25+ cigarettes compared to 1 in nonsmokers. In females, the corresponding ratios were 10.5, 28.5 and 32.0 respectively.

Mortality from COLD in pipe and cigar smokers is also higher compared to nonsmokers but the risk is less than that for cigarette smokers (1).

Risk associated with smoking : studies in India

The prevalence of chronic bronchitis in male smokers was 20% and in male nonsmokers 5.19% in a study in Chandigarh (15),giving a 4-fold risk in smokers. The age-adjusted risk ratio in smokers for the same data is 3.75. Other studies also show similar risk ratios in smokers for chronic bronchitis. No data on risk of mortality in smokers is available.

From the study of Malik (14) the estimated risk of chronic bronchitis in cigarette smokers is 3.3, in bidi smokers,3.9 and in hukka smokers,9.7.

Proportion of COLD attributable to smoking

For estimating the porportion of COLD in the population attributable to smoking, estimates for proportion of smokers in the population besides risk of COLD in smokers is required. Various studies report regional and social class variation in proportion of males who smoke (36, 37). However, an average estimate for the country can be deduced from the bidi and cigarette tobacco cleared annually for domestic consumption and an assumed average frequency of smoking of 15 bidis per day or 10 cigarettes per day[+]. Furthermore, by considering 95% of smokers as males,the proportion of smokers works out to about 50% in males and 2.5% in females.

With a 4-fold risk of COLD in smokers and 50% of the male adult population and 2.5% adult females as smokers, the proportion of cases attributable to smoking works out to 60% in males and 7% in females. Assuming similar risk of mortality due to COLD in smokers, 60% of the COLD deaths in males and 7% in females are due to smoking.

Thus, between 7 and 13 million of prevalent COLD cases and 0.11 to 0.21 million COLD deaths are attributable to smoking and therefore avoidable.

Discussion and conclusion

Prevalence of COLD reported from surveys in India show a wide variation ranging from 0.7% to 12.5% (17). Mortality rates are available for Bombay city and can also be computed from the data obtained in a rural survey on causes of death (22). The crude mortality rate for COLD in Bombay was 0.5 per 1000 for adults, (and 0.3 per 1000 in the population). The corresponding rate in the rural survey was 1.3 per 1000 (and 1.2 per 1000 in the population). Both these rates have to be viewed with reservation. The Bombay rate is reliable but not applicable to the

[+] In 1981, 130 million kg and 83 million kg were cleared for bidis and cigarettes respectively (38). This works out to 500,000 million bidis and 86,800 million cigarettes annually. With the above given frequency of smoking, there would be 91 million bidi smokers and 24 million cigarette smokers, giving a total of 115 million smokers.

country due to regional variation in prevalence of COLD whereas the survey rate may not be reliable due to lack of accuracy in assigning the cause of death in rural areas.

With the limited data available estimates for morbidity and mortality due to COLD in the country are estimated to be between 15 and 29 million and 0.25 to 0.46 million, respectively. Thus, in adults the prevalence is likely to be in the range of 3.2 to 6.1 percent and mortality in the range of 0.5 – 1.0 per 1000. Furthermore, between 7 and 13 million prevalent cases and 0.11 to 0.21 million COLD deaths are attributable to smoking and therefore avoidable. Needless to state that these estimates may need revision when more data accumulates on the distribution of the disease in the country.

Smokers in India, who smoke mostly bidis, are at as much risk due to COLD as cigarette smokers. Higher risk is reported for hukkah smokers, although emission levels are lower than in a bidi. However, hookah smoking may not be a serious problem as it is losing in popularity.

It may be mentioned here that in all the discussion of Indian data, chronic bronchitis and COLD are used interchangeably.

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Tobacco Use in Pregnancy and Reproductive Outcome

S. Krishnamurthy[1]

Studies of maternal use of tobácco during pregnancy and associated reproductive outcome are reviewed. Maternal tobacco use impacts directly and indirectly on the developing foetus, newborn, infant and child. Direct effects relate to tobacco use during pregnancy. Indirect effects relate to maternal tobacco use in general. Western data strongly implicate maternal smoking in pregnancy with increased risks of low birthweight, spontaneous abortion, stillbirths, foetal deaths, and neonatal deaths. Women in India mainly chew or apply powdered tobacco. Indian data show a three-fold increase in stillbirths and a 100-400g decrease in birthweights associated with maternal smokeless tobacco use during pregnancy. Other associations are : an average increase in placental weight of 66g, and increased male foetus wastage. A two-fold increase in perinatal mortality was observed in biri smokers' newborns in Bangladesh. Maternal tobacco use has reportedly synergistic effect alongwith maternal and neonatal anaemia, vitamin and nutritional deficiencies and inadequate antenatal and paediatric health care, on perinatal morbidity.

Population surveys from four different places in India have shown that 10–60% of 15–44 year old women chew tobacco. Estimates derived from tobacco consumption in India suggest that about 10% adult women chew tobacco, and 2.5% smoke bidi. The population impact of such tobacco use and therefore of potentially preventable adverse reproductive outcome is estimated to be approximately 17% of stillbirths, and about 11–12% of perinatal mortality. Impact on low birthweight is not estimable from the published material. Besides these direct effects of maternal tobacco use in pregnancy, indirect effects of such use are infantile and childhood morbidity and mortality due to low birthweight and prematurity. Malnutrition due to diversion of scanty purchasing power from nutrition to tobacco has been documented.

Finally other associations with maternal or parental cigarette smoking are reviewed. They include sudden infant death syndrome, nicotine and cotinine transfer via breast milk and excretion by the neonate in saliva and urine, infantile bronchitis and pneumonia, persistent middle-ear effusions, and impaired physical, mental, and emotional development. Although these associations have so far been documented only with parental cigarette smoking it may not be unlikely to expect similar associations with parental bidi smoking and even smokeless tobacco use.

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Introduction

This paper addresses the effects of maternal tobacco use on the unborn foetus, on pregnancy outcome in general, and on the developing infant and child. After a brief review of information from Western studies, available Indian data is examined and the population burden of health problems associated with maternal tobacco use, assessed.

Tobacco use and reproductive outcome

Western studies

The adverse effects of maternal cigarette smoking in pregnancy on the unborn foetus, newborn offspring and antenatal events including antepartum haemorrhage and abruptio placentae, are well documented (1–4) in the West.

A number of studies have confirmed Simpson's observation (2) that babies born to women who smoke cigarettes during pregnancy, are on an average 200g lighter – in fact almost twice the number of these newborns weigh less than 2500g compared to newborns of mothers who do not smoke (1). A dose-response relationship has also been established i.e. with increase in maternal smoking during pregnancy, a corresponding decrease is observed in baby's birthweight (1). The risks of spontaneous abortion, foetal deaths, stillbirths, and neonatal deaths are also increased with maternal smoking (1). Perinatal mortality rates are also increased (3). These effects are independent of other factors affecting birthweight (1).

Placental weight increases have also been reported and are dose-related to maternal tobacco use. Furthermore, all parameters of foetal growth viz., head and chest circumferences, body length etc., are reduced in babies of women who smoke. The child's development upto 11 years is adversely affected by maternal smoking in pregnancy (1). Synergism with factors like anaemia in pregnancy and increased parity has been mentioned (3).

Mechanisms of the tobacco smoking effect are thought to include foetal anoxiemia associated with carbon monoxide and the vasoconstrictive action of nicotine on the foeto-placental circulation (5). More research is needed in this area.

Studies in India

Tobacco use by women in India : Methods of tobacco use by women in India differ from those in the West. Many forms of tobacco use exist in different parts of the country. They include different ways of chewing, smoking and applying tobacco. When chewed it is used alone, or mixed with lime, or as a mixture with betel nut and lime, with or without betel leaves, with camphor, cloves and other spices. It is also smoked in several ways. The most frequent way is termed a 'bidi' or 'biri' in Bangladesh. The bidi is a hand-rolled cigarette-like structure using uncured tobacco wrapped in a tendu leaf. Another method of smoking is as a hand-rolled cigar called "chutta" with the lighted end in the mouth (mainly in Andhra

Pradesh); or as a hookah with a water filter; or in clay pipes, etc. Powdered burnt tobacco is also applied to the gums and teeth as "masheri" or "mishri", and used as a tooth paste/tooth powder. Finally it may be taken nasally as snuff. It may well be used in myriad other, as yet undescribed ways. However, the Indian woman more often chews or applies burnt tobacco orally, than smokes.

Prevalence of tobacco use in 15–44 year-old women in India : The actual prevalence of tobacco use in reproductive age women in India is not known. However, data from different studies focussing on specific groups, are available. In one, the number of female tobacco users in the population is estimated from house-to-house surveys in four different rural parts of India (6). Specific information on 15–44 year old women suggests tobacco users range from 10% to 60% in these areas. Since these four study areas are known for the high prevalence of oral cancer and precancer, the actual tobacco chewer and smoker prevalence rates in reproductive age women in the rest of the country may be lower than derived from this study–specific population. Another study of patients in a large teaching maternity hospital showed 33% of 500 women used tobacco in any form (7). These data too are subject to selection bias.

The estimate of prevalence of tobacco use in the adult Indian female,based on the available data of annual consumption of tobacco in the country (8), may be the best available estimate. These estimates are that about 10% adult females are chewers and 2.5% are smokers. Projecting from 1981 census data, this suggests about 16.4 million adult Indian women were tobacco chewers and 4.1 million were smokers in 1986.

Indian studies of maternal tobacco use and reproductive outcome : The effect of maternal tobacco use on reproduction in India is of special interest since carbon monoxide and tar are not products of smokeless (chewed and applied) tobacco, as they are of smoked tobacco. The mechanism of an identifiable adverse outcome of smokeless tobacco use will likely be different from smoking tobacco. Comparisons of the effects of maternal smoking and use of smokeless tobacco on pregnancy outcome may help shed light on the mechanism of such adverse effects.

Three studies in Pune and Jabalpur (9–11) report associations between maternal smokeless tobacco use and adverse reproductive outcome. An additional study in Bombay conducted after this paper was presented in April 1987, has yielded similar results (7). Findings from these data are listed in Table 1 and described below.

(a) Stillbirth : The first report (9), from a Pune hospital, is of 1388 singleton births without gross congenital abnormality. Of these, 220 births occurred to women who chewed tobacco and 1168 to those who did not use tobacco in any form. There were 11 stillbirths in the exposed group and 20 stillbirths in the unexposed group. Thus the crude stillbirth rate per 1000 births was 50.0 in women who chewed tobacco compared to a rate of 17.1 in women who did not use tobacco, yielding an approximately 3- fold relative risk.

Table 1. Adverse reproductive outcome and maternal tobacco use in pregnancy–Indian data*

Reproductive parameter year & reference	Findings: Tobacco users	Findings: Tobacco nonusers	Method of tobacco use
Still births			
1978 (9)	50.0/1000 births	17.1/1000 births	Chew
Low birthweight			
1978 (9)	100–200 g less+		Chew
1983 (11)	Average 395 g less+		Chew & apply
Unpublished (7)	42/65 liveborns	41/113 liveborns	Apply masheri
New born male : female ratio			
1978 (9)	0.8	1.09	Chew
Unpublished (7)	0.8	1.06	Apply masheri
Placental weight			
1983 (10)	66 g more+		Chew & apply
Perinatal mortality			
1981 (12)	270/1000 births	122/1000 births	Smoke biris

* Except for information on perinatal mortality, which is from Bangladesh

\+ compared to nonusers'

(b) Low birthweight : A decrease in birthweight ranging from 100 to 200g in the above mentioned study (9), and upto 400g in another (10) was noted in newborns of women with tobacco habits compared to newborns of women without the habit. In the Bombay study (7) also, a significantly higher proportion (0.65) of low birthweight (< 2.5 kg) babies (single liveborns) were born to women who used tobacco during pregnancy compared to those who did not (0.36)

(c) Increased male foetus loss : Fewer male foetuses were born to women who chewed tobacco (80 males per 100 females) compared to women who did not (109

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males per 100 females) (9). Similarly women who used tobacco mostly as masheri had fewer male newborns (81 per 100 females) compared to women who did not (106 males per 100 females) (7).

(d) Increased placental weights : Placental weights were on an average 66g more in tobacco chewers' pregnancies than in nonchewers' (11). The association was statistically significant when tobacco was chewed regularly for more than six years. No information was available on the ratio of placental weight to birthweight.

(e) Increased perinatal mortality : Although data are not available from India, there are reports (3, 12) from Bangladesh associating maternal "biri" smoking with a 2-fold increase in perinatal mortality. In Bangladesh, women smoke tobacco as a handrolled cigarette using uncured tobacco. This is called "biri" and is similar to the Indian "bidi".

Assessment of adverse effects attributable to maternal tobacco use

It is worthwhile to assess the impact that preventing tobacco use in reproductive age women may have on reducing adverse reproductive outcome. The measure used for this assessment is the Population Attributable Risk (PAR). The proportion of the outcome that would be prevented if tobacco was not used is expressed as a percentage. The PAR% can be determined from the prevalence (P) of the exposure factor in the population and the relative risk (R) of outcome in those exposed compared to those not exposed and is expressed as $P(R-1)/(P(R-1)+1)\%$.

Load of stillbirths

The estimated number of women aged 15–44 years is 164 million in the country (1986 population) from 1981 census projections (13). Furthermore, at the reported (13) rate of 140 livebirths per 1000 women in the age group 15–44 years, there would be 23 million livebirths in the country.

Information for Bombay city (1981 population) as published by its municipal corporation (14) indicates that there are 30 stillbirths per 1000 livebirths. Assuming this rate is applicable to the country, approximately 700,000 stillbirths would occur to women in the reproductive age group in the population.

The reported 3-fold excess risk of stillborn infants to women who chew tobacco (9), and a 10% prevalence of this habit in the females of the population, would yield a PAR of 17%. Thus approximately 119,000 stillbirths in the population would be attributable to maternal tobacco chewing during pregnancy and hence preventable if tobacco was not thus used.

Low birthweight (LBW) load

Relative risk estimates of low birthweight babies born to women who chew

tobacco or smoke bidis are not available from published reports. It is therefore not possible to estimate the proportion of low birthweight babies attributable to tobacco use.

Increased placental weight

Published information regarding the risk of placental pathology associated with tobacco use–both smoked and smokeless – in pregnancy is meagre. Estimates of the population burden of this problem and the proportion attributable to maternal tobacco use cannot be arrived at the present time.

Decreased male : female ratio in newborns

Maternal cigarette smoking (15), tobacco chewing (9), and oral application of burnt tobacco (7), in pregnancy are reportedly associated with decreased male : female (M : F) ratio of their offspring compared to the ratio in nonusers' offspring. The M : F ratio of cigarette smokers' babies was 0.97 compared to 1.07 of nonsmokers' babies (15). The M : F ratios were 0.8 in newborns of tobacco chewers versus 1.09 in those of women who did not chew tobacco (9). Recent, as yet unpublished data from a leading maternity hospital in Bombay yielded M : F ratios of 0.8 in tobacco users' newborns compared to 1.06 in those of women not using tobacco (7). This observation needs to be confirmed, before making any assessments.

Increased perinatal mortality

A 2-fold increase in perinatal mortality of babies born to women who smoke 'biri' as compared to those who do not has been reported from Bangladesh (3, 12). If the same risk is assumed for Indian women who smoke bidis; a 2.4% of perinatal mortality would be attributable to maternal smoking.

No information is available on the association of perinatal mortality with maternal tobacco chewing. However, it may not be unreasonable to assume the same 2-fold risk. The rationale being that two-thirds of perinatal mortality reportedly (16) occurs in LBW babies, which in turn occurs more frequently in women who use smokeless tobacco compared to those who do not. If so, an estimated 9% of perinatal mortality would be attributable to maternal tobacco chewing.

At a perinatal mortality rate of 53 per 1000 births in the country (17), an estimated 1,256,000 perinatal deaths would occur in 1986 of which about 143,000 would be attributable to maternal tobacco use.

Other effects of maternal tobacco use

Other adverse effects of maternal tobacco use on the newborn could include prematurity, its related morbidity and mortality from infectious diseases like measles, bronchitis, pneumonia, tuberculosis etc. and from failure to thrive. Such

morbidity in infancy can also have major longterm effects on the growing child.

Parental tobacco use may indirectly cause malnutrition in children of poorer sections of society. Data cited from Bangladesh (3, 12) have shown that socio-economically deprived people are greater users of tobacco than those belonging to high income bracket. Upto 50% of a poor person's food purchasing economic power can be diverted to tobacco purchase, resulting in malnutrition due to inadequate food purchasing ability. Upto 2.5% of under-five children in Bangladesh may have severe protein-calorie malnutrition.

Several other adverse effects have been reported in the literature with respect to maternal cigarette smoking. These are cited below for completeness of information. Published reports of associations of these conditions with maternal bidi smoking or smokeless tobacco use, are not available.

(a) The breast fed infant of a smoking mother is reported to consume nicotine and cotinine in the breast milk (18, 19) and also to excrete these toxic substances in the saliva and urine (19).

(b) A positive association has been found between maternal cigarette smoking and sudden infant death syndrome (SIDS) in several studies cited in the U.S. Surgeon-General's report (1).

(c) Persistent middle-ear effusions occur nearly three times as often (RR: 2.8, CI: 1.1 – 7.0) in infants exposed to parental smoking and household tobacco smoke compared to infants not so exposed (20).

(d) Infantile bronchitis and bronchopneumonia have been found to be associated with parental smoking habits in a study of 2077 infants, cited by Coleman et al (3).

Discussion

In short, published Indian data (9 – 11) have shown a three- fold increase in stillbirths, a decrease in birthweight ranging from 100 to 400g, an increase in male foetus wastage, and an average 66g increase in placental weight associated with maternal smokeless tobacco use during pregnancy. A two-fold increase in perinatal mortality in biri smokers' newborns was reported from Bangladesh (3, 12).

There is very little information on the mechanism of effects of maternal smokeless tobacco use on the unborn foetus and the newborn. Smokeless tobacco use does not generate carbon monoxide, tar, and polycyclic hydrocarbons as does smoked tobacco. Therefore, the noxa to the unborn foetus from smokeless tobacco use is most likely due to nicotine-mediated placental vasoconstriction and resultant foetal anoxiemia. Other toxic substances in chewing tobacco and masheri – burnt tobacco used in oral application – may also be involved. The nature of such toxic substances is as yet speculative although information on the mutagenic effects of nitrosamines in chewing tobacco is presented elsewhere in this volume (21).

Even though the actual mechanism of damage to the foetus is unclear, data regarding both smoked and smokeless tobacco use show that adverse foetal outcome is associated with such use in pregnancy. It is reported that the male foetus is more vulnerable to adverse influences in pregnancy than the female (4). The increased male foetus wastage associated with cigarette smoking (15), tobacco chewing (9), and masheri application (7), by women during pregnancy needs to be confirmed in larger studies. This finding, if confirmed, may provide a potent tool for control of maternal tobacco use during pregnancy in particular, and by women in the reproductive ages in general, since Indian society places such a high premium on a male child. Publicising these observations may help in preventing tobacco use by those at risk.

The transmission of cotinine and nicotine via breast milk to the breast fed infant of a cigarette smoking mother (18, 19) may affect the baby physiologically. Such physiologic nicotine stimulation may be the beginning of nicotine dependence. Similarly the bidi smoking mother's newborn breast fed infant may become nicotine dependent. Data are needed to explore such a possibility. Investigation is also needed on a similar association with maternal smokeless tobacco use during lactation and breast feeding.

Premature birth with attendant low birthweight are the most frequent causes of death of infants in the first year of life in urban and rural India (13). The two-fold increase in perinatal mortality in Bangladeshi "biri" smokers' offspring (3, 12) may also, in part, be an indirect effect of maternal smoking on the newborns' weight and ability to thrive. Maternal tobacco use has synergistic effect alongwith neonatal anemia (3) vitamin and nutritional deficiencies (3, 12), and inadequate paediatric and antenatal health care (3), on perinatal morbidity.

Finally, it may well be that a proportion of the high infant mortality in the country may be consequent on prematurity, low birthweight and its indirect effects on the infant's ability to withstand other noxae. Thus a fraction of these deaths may be indirectly attributed to maternal tobacco use in pregnancy.

The western associations of parental or maternal smoking with SIDS (1), persistent middle ear effusions (20), infantile bronchitis, pneumonia (3) and impaired development (1) need studying in the context of bidi smoking and smokeless tobacco use in India. Little is known at present regarding other adverse effects of passive exposure of the under-five child to such tobacco use.

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An Assessment of Excess Mortality Caused by Tobacco Usage in India

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It is well known that cigarette smokers experience a significantly higher mortality compared to non-smokers. In India, cigarette smoking forms only a minor percentage of overall tobacco usage and therefore, western results cannot be extrapolated. Three reports on mortality experience of tobacco users (including chewers and smokers) in India are available and they all show that tobacco users age 15 years and over experienced a significantly higher mortality compared to nonusers. The relative risk for overall mortality ranged between 1.3 and 1.9 for men and women. Percentage of people using tobacco in the general population is available from house-to-house studies in six different parts of India and it ranges from 44% to 74%. Among men tobacco is used by 60% to 80% but among women there is a wide variation, between 15% to 67%. It can be thus estimated that among men at least 19% and upto 40% and among women at least 4% of all deaths are caused by tobacco. In India among individuals age 15 years and over, about 2.76 million deaths occur among men and 2.3 million among women. Total number of deaths caused by tobacco by all known and unknown causes are thus estimated as at least 6,30,000, possibly higher, upto a million. These findings emphasize the fact that tobacco usage is a serious health problem in India right now and would become worse unless remedial actions are initiated.

Introduction

It is well known that cigarette smokers experience a significantly higher mortality compared to non-smokers. Major reasons for this excess mortality are cardiovascular diseases, lung cancer, respiratory diseases and other tobacco related cancers.

The relationship between lung cancer and cigarette smoking was postulated through case-control studies and later reconfirmed by prospective studies. These prospective studies showed that cigarette smokers experienced a significantly higher mortality compared to non-smokers. Because of the availability of an efficient and reliable death registration and certification system in industrialized countries, it was possible to obtain reasonably accurate information on the cause of death and study specific reasons for excess mortality.

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In India, the usage of tobacco is not restricted to cigarette smoking alone. It is smoked in a variety of ways like bidi, chutta, claypipes, hookah, dhumti etc. It is chewed as an ingredient of betel-quid, by itself, mixed with lime, mixed with lime and areca nut etc. It is applied in the form of mishri, gudakhu, paste etc. Almost all these forms of tobacco usage are shown to be associated with oral cancer and oral precancerous lesions. In addition, bidi smoking is shown to be associated with cancer of the pharynx, larynx and esophagus, cardiovascular diseases, and respiratory diseases.

Prospective studies of these forms of tobacco usages have been conducted mainly for studying their relationship with oral cancer and oral precancerous lesions. Report on overall mortality rates are available from three of these studies. There is no comprehensive death registration system in rural areas where these studies were conducted, so specific causes of mortality were unknown.

Mortality in tobacco users

In the first study a group of smokers, (almost all bidi smokers) and group of chewers (almost all tobacco-lime chewers) were followed for 8 years in rural areas of Pune district. Overall mortality was higher among smokers compared to chewers. Age-adjusted relative risk (of overall mortality) among smokers compared to chewers was 1.6 (1).

In a study in Ernakulam district of Kerala, a random sample of 10,287 individuals age 15 years and over was followed over a period of 10 years (2). The age-adjusted relative risk (of overall mortality) among tobacco users compared to non-users was 1.4 among men and 1.3 among women. These relative risks were highly significant. Also they were much lower than crude relative risks (2.5 and 3.4 respectively) because tobacco users were concentrated more often in the higher age groups compared to non-users. Among smokers (mostly bidi smokers) age-adjusted relative risk was 1.5 and among chewers (mostly betel quid chewers) it was 1.3, and both were significant. These excess risks were not explained by oral cancer alone, and therefore this study, for the first time, raised the possibility that tobacco chewers may experience a significantly higher mortality from causes other than oral cancer that are yet unknown.

In Srikakulam district of Andhra Pradesh, a random sample of 10,169 individuals age 15 years and above was followed up for 10 years (3). The most common tobacco habit was reverse chutta smoking i.e. smoking of chutta with the lighted end inside the mouth. The relative risk of overall mortality of this form of smoking was 1.9 for men as well as women. The relative risk for chewing was also 1.9 and for conventional smoking which included bidi and chutta smoking, it was 1.8. Thus relative risks were much higher in Srikakulam compared to Ernakulam.

The two areas, Ernakulam and Srikakulam defer considerably. In Ernakulam, literacy rates are quite high, good health care facilities are available, and population mortality rates are low. Bidis used in Ernakulam are generally shorter

in length (about 4 cm.) compared to other places in India (5–7 cm.) and contain smaller amount of tobacco. In contrast, in Srikakulam district the literacy is low, health care facilities are not very adequate and population mortality rates are high. Tobacco is mostly smoked in the form of cheroot which is simply a roll of unprocessed tobacco leaf. Thus it is possible that the relative risks obtained in Ernakulam are on somewhat lower side compared to the risk in general and those obtained from Srikakulam are on somewhat higher side. The range of relative risk of overall mortality is between 1.3 and 1.9, variation being higher among women than among men.

Prevalence of tobacco usage in India

It is a well known fact in epidemiology that given the relative risk of overall mortality for a risk factor and the proportion of population exposed to that risk factor, it is possible to estimate the proportion of total mortality that is attributable to, or caused by, the risk factor. (If R is the relative risk and P is the proportion of the population exposed to the risk factor such a proportion is given by the expression $P(R-1)/(1 + P(R-1))$. Since we already have an estimate of relative risk, in order to estimate the proportion of mortality in the population attributable to tobacco usage, we need to know the percentage of tobacco users in India. This information is available from 5 areas of India based on random samples of 10,000 individuals age 15 years and over in each area (4), from one area based on a sample of 1,00,000 individuals age 15 years and over (5), and from one area based on a sample of 35000 individuals age 35 years and over (6). The percentage of tobacco users in these studies ranged from 44% to 74% (table 1). Among women the prevalence of tobacco usage ranges from low of 15% to a high of 67%. Among men the tobacco usage is more consistent, ranging from 62% to 82%.

Table 1. Prevalence of tobacco usage in India from population based house-to-house studies.

Area	Sample Size	Tobacco users		
		Men	Women	Over all
Bhavnagar	10,071	71%	15%	44%
Ernakulam	10,287	81%	39%	59%
Srikakulam	10,169	81%	67%	74%
Singbhum	10,048	81%	33%	56%
Darbhanga	10,340	78%	51%	64%
Pune	1,01,761	62%	49%	64%
Mainpuri	34,997	82%	21%	57%

Table 2 shows breakdown of tobacco habits in terms of chewing, smoking and mixed habits for men and women. A considerable amount of variation is apparent here. In Pune, over half of menfolk chew tobacco whereas chewing habits are rare in Bhavnagar and Srikakulam. Major chewing habits were betel quid chewing in Ernakulam, and tobacco-lime, often combined with areca nut, in all other areas. Number of individuals chewing regularly but without tobacco was very small (less than 1%) in all these areas.

Smoking habits were rare in Pune but in all other areas, over half of menfolk (including mixed habitues) smoked. The most common smoking habit was bidi smoking in all areas. Cigarettes were smoked by 5% men in Ernakulam and by less than 1% men in other areas. Other common smoking habits were claypipe in Bhavnagar, and chutta in conventional and reverse manner, in Srikakulam.

Table 2. Different type of tobacco usage in India

Area	Men			Women		
	Chew	Smoke	Mixed	Chew	Smoke	Mixed
Bhavnagar	9%	56%	6%	15%		
Ernakulam	14%	45%	22%	38%	1%	1%
Srikakulam	4%	70%	7%	3%	64%	–
Singbhum	17%	50%	14%	26%	5%	2%
Darbhanga	28%	24%	26%	7%	41%	4%
Pune	53%	6%	2%	49%	–	–
Mainpuri	21%	41%	20%	9%	11%	1%

Chewing habits among women are low in Srikakulam and Darbhanga but smoking habits among women are high in these two areas. Women in Srikakulam smoke chutta, almost always in reverse fashion, and women in Darbhanga smoke hookah. A large percentage of women in Pune apply mishri, a kind of roasted and powdered tobacco, and women in Ernakulam chew betel-quid with tobacco.

It is clear that there is a wide variation in the type of tobacco usage as well as in the prevalence of different types of tobacco usage. We do not have information regarding excess mortality associated with each of the different type of tobacco usage. Under the circumstances it seems reasonable to work with overall relative risk of mortality among tobacco users and overall prevalence of tobacco use among men and women, realising that variations are much greater among women than among men.

Mortality attributable to tobacco usage

Given these percentages and the relative risk of overall mortality, it is possible to estimate what percentage of total deaths are attributable to the usage of tobacco. Table 3 shows the results of such calculations for men, and Table 4 for women. It shows that even with most conservative estimates, 19% of all deaths among men and 4% among women can be attributed to the usage of tobacco. Using estimates from Srikakulam district, about 40% or two-fifths of all deaths can be attributed to the use of tobacco among men as well as women.

Table 3. Percentage and number of deaths attributable to tobacco usage among men in India.*

Relative risk	Tobacco usage 60%	70%	80%
1.4	19% (5,30,000)	22% (6,00,0000	24% (6,70,000)
1.5	23% (6,40,000)	26% (7,10,000)	29% (7,90,000)
1.7	30% (8,20,000)	33% (9,10,000)	36% (9,90,000)
1.9	35% (9,70,000)	39% (10,70,000)	42% (11,50,000)

* Estimated total deaths among men age 15 years and over 2.76 million.

Table 4. Percentage and number of total deaths attributable to tobacco usage among women. *

Relative risk	Tobacco usage 15%	30%	45%	60%
1.3	4% (99,000)	8% (1,90,000)	12% (2,70,000)	15% (3,50,000)
1.4	6% (1,30,000)	11% (2,50,000)	15% (3,30,000)	19% (4,50,000)
1.5	7% (1,60,000)	13% (3,00,000)	18% (4,20,000)	23% (5,30,000)

* Estimated deaths among women 15 years and over 2.3 million.

On the basis of projected population figures for 1986 and latest available age-specific death rates it can be estimated that among individuals age 15 years and over annually about 2.76 million deaths occur among men and 2.3 million among women. Applying minimum estimates it would seem at least about 6,30,000 of these deaths annually are directly attributable to the use of tobacco. Considering whole of India, minimum estimate for men is probably on a lower side and true figure may be higher. Among women, variations are so wide, and the amount of available information so little, that it may be prudent to stick to the minimum estimate. Thus we can say that the number of deaths attributable to tobacco usage in India is in the range of 6,30,000 to about a million per year.

One must point out that this estimate is on the basis of age-adjusted relative risks i.e. by artificially enforcing tobacco users and non-users to have similar age distribution. In reality, tobacco users are more concentrated in higher age-groups. Therefore the actual number of deaths attributable to tobacco usage, at present, would be higher. Also the estimates of tobacco usage although best among all available ones, are over 20 years old. Tobacco usage has, most probably increased during this period.

These calculations show that the health consequences of tobacco usage is a very serious problem in India right now and it is contributing to more than 6,30,000, possibly up to a million, extra deaths every year. It is thus a present and not a future problem. In future it could become an even more serious problem unless remedial measures are initiated. Smoking habits which show higher relative risk for overall mortality are known to be spreading very rapidly in the younger age groups.

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Assessment of Morbidity and Mortality Due to Tobacco Usage in India

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Introduction

The evaluation of hazards associated with tobacco usage in human populations has been actively pursued over the world, for well nigh over a quarter century. The first comprehensive report on hazards of cigarette smoking appeared in 1964 (1) followed by several others (2, 3) implicating cigarette smoking to be a risk factor for cancer of certain sites, cardiovascular disease (CVD) and chronic obstructive lung disease (COLD).

The objective of this paper is to provide a general background with respect to tobacco practices prevalent in India together with brief information on toxic components of the local products and the risks associated with its use as documented by several epidemiololgical studies carried out in the country and finally to assess from this limited data the overall load due to these practices.

Tobacco practices and product evaluation with respect to toxic constituents

India ranks 3rd in the world tobacco production and over 80% of tobacco cleared for home consumption (excluding agriculture and industrial use) is smoked. Bidi (obtained by wrapping 0.2 g to 0.3 g of tobacco in tendu leaf) is the most popular form, though other forms of smoking like chilum (clay pipe) chutta (kind of cigar) and hookah are prevalent in certain regional pockets. Tobacco is also chewed either as an ingredient of betel quid, or mixed with lime etc. Dental application of burnt tobacco (masheri) is also practiced. Recently, tooth pastes called "creamy snuff", with tobacco as the major ingredient, are being marketed in the country, under various brand names.

The overall toxicity, as measured by total particulate matter (tar) and nicotine, showed that bidi and cigarette have high levels of tar (23–41 mg per bidi and 19–28 mg per cigarette) and nicotine (1.7–2.8 mg per bidi and 0.9–1.8 mg per cigarette) which in turn have higher levels of these noxious components than the

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Abbreviations : CVD : Cardiovascular disease, CHD : Coronary heart disease, MI : Myocardial infarct, COLD : Chronic obstructive lung disease.

cigarettes currently marketed in the West (4,5).The other major components of smoke considered to be harmful are (a) carbon monoxide which is considered to interfere with tissue oxygenation and is reported (4) to be as high as 7.7 vol % in bidis and 3.5 vol % in western cigarettes, (b) hydrogen cyanide, a strong cilia toxic agent is reported (6) to vary from 688 μg to 904 μg per bidi and 366–638 μg per cigarette, (c) phenols, which are tumor promoters,vary from 129 μg to 273 μg per bidi compared to 118–226 μg per cigarette (6) and (d) benzo(a)pyrene, a known carcinogen, varies from 108–144 ng per bidi and 85–114 ng per cigarette (6).

In chewing tobacco, several N-nitroso compounds, which are potent carcinogens have also been identified (7).

Risks associated with tobacco habits for various diseases and overall mortality (Indian studies)

Overall mortality

Reports (8–10) on mortality experience of tobacco users have shown that they experience a significantly higher mortality compared to nonusers. The age-adjusted relative risks of overall mortality ranged between 1.1 and 2-fold as shown in Table 1. It is seen that bidi smokers have equally high mortality risk ratios as cigarette smokers (10).

Table 1. Estimates of mortality risk ratios in tobacco users[+] (Indian studies)

Relative Risk (RR) of overall mortality in	Kerala (Ernakulum) (Age ⩾ 15 yrs)	Andhra (Srikakulum) (Age ⩾ 15 yrs)	Bombay (Blue collar workers) (males 40–59 yrs.)
Tobacco user	1.4 (M) 1.3 (F)		
Smoker	1.5 (M) 1.1 (F)	1.8 (M)	2.2 (Bidi) 2.1 (Cigarette)
Chewer	1.2 (M) 1.3 (F)	2.0 (M)	
Reverse smoker		1.9 (M) 1.9 (F)	

[+] Compared to nonusers of tobacco. M: Male, F: Female.

A study in rural Maharashtra (11) which undertook follow-up of leukoplakia cases and matched healthy controls, for a period of 8 years, has reported 60% higher mortality in smokers compared to chewers of the healthy control group.

and in the leukoplakia group a 2-fold higher mortality in smokers as compared to chewers.

Major tobacco related cancers

Both tobacco smoking and chewing have been implicated in cancers of the oral cavity, pharynx, larynx and oesophagus (12, 13) and bidi and cigarette smoking in cancer of the lung (14, 15). The associated risks for various sites, from 2 studies (13, 15) are shown in Table 2.

Table 2. Estimates of cancer incident risk ratios (RR) in tobacco users[+] (Indian studies)

Site	Chewer	Smoker	Chewer & smoker
Oral cavity	6.0	2.8	10.1
Oropharynx	3.3	11.8	31.7
Hypopharynx	6.2	3.6	16.9
Larynx	4.6	7.7	20.1
Oesophagus	2.5	2.2	6.2
Lung	–	14.9 (bidi) 10.2 (cigarette)	–

[+] Compared to nonsmoker-nonchewer.

In a cohort study in Bombay, risk of bidi and cigarette smokers for developing upper alimentary and respiratory tract cancers has been estimated to range between 5.2 and 6.2 compared to the nonexposed group, in the blue collar workers (10).

Cancer mortality risks are not available.

Cardiovascular disease (CVD)

Several epidemiological studies associating smoking with coronary heart disease (CHD) have been reported in the country (16, 17). Risks of smokers for developing CHD and in particular, myocardial infarct (MI) have been assessed in two studies, one is a case-control study (18) and the other a cohort study (10). The estimated risks were observed to be broadly similar for bidi and cigarette smokers and varied from 2 to 3-fold as seen in Table 3.

Furthermore, there is epidemiological evidence to suggest that bidi and cigarette smoke have equally adverse effects on some of the parameters known to be associated with the pathogenesis of the disease such as increasing pulse rate

Table 3. Estimates of CHD risk ratios (RR) in smokers[+] (Indian studies)

	Bidi Smoker		Cigarette Smoker	
	CHD	MI	CHD	MI
Case-control study	3.1	3.8	2.5	3.3
Cohort of blue collar workers	3.3	3.0	3.0	2.8
Cohort of white collar workers	–	–	1.1	1.6

[+] Compared to nonsmoker-nonchewer

and blood pressure (19), and also increase in platelet aggregation, serum free fatty acid levels and blood sugar levels (20, 21).

CHD/MI mortality risk ratios are not available. Incidence risk ratios and mortality risk ratios are also not available for the other two CVD conditions known to be associated with smoking viz., cerebrovascular disease and peripheral vascular disease.

Chronic obstructive lung disease (COLD)

A study (22) conducted in northwest India estimates the risk of chronic bronchitis in cigarette smokers as 3.3, bidi smokers as 3.9 and hookah smokers as 9.7. Another study (23) from the same area also shows similar risk ratio (age-adjusted) for chronic bronchitis in smokers (3.75). Risk for emphysema is not reported.

No data on mortality risk ratios are available.

Other conditions

The deleterious effects of maternal tobacco use on the unborn foetus, the newborn offspring, and antenatal events have been reported from the west. In India, there are a few studies (24, 25) documenting effect of maternal chewing viz., decrease in birth weight, increase in mortality in utero (stillbirth), increased loss of male infants at birth, and perinatal mortality.

Risk for giving birth to a stillborn offspring in women who chew tobacco can be computed from the available data (24) to be 3-fold. Risks for other conditions are not available.

Estimates of prevalence of different tobacco habits in the country

In absence of a national sample survey on tobacco usage, authentic estimates of prevalence are not available. However, in 7 selected areas with high prevalence of oral cancer, sample surveys have shown that the prevalence of chewing varied

from 11% to 55% in males and 10% to 39% in females (26). Prevalence of smoking varied from 8% to 77% in males but was not common in females (2% – 12%) except in some regional pockets where traditional forms of smoking were practiced (chutta smoking in Srikakulum and hookah smoking in Darbhanga) (26). Bias in selection of areas and the large regional variation do not justify extrapolation of these prevalence rates to the country. Even so, this data is useful in fixing bounds for prevalence of tobacco usage.

Another approach is to estimate prevalence of smoking and chewing habits from annual tobacco consumption data of the country.

In 1981, annual consumption of bidis and cigarettes was reported to be 500,000 million and 86,800 million sticks respectively (27), i.e. daily consumption of 1,370 million bidis and 238 million cigarettes respectively. If on an average an individual smokes 15 bidis or 10 cigarettes per day, then there would be 115 million smokers in the adult population. If all the 115 million smokers were males then the prevalence of smokers in males would be 54.6% and of course zero in females. However, a more reasonable assumption of 5% of smokers to be females (95% males) would give prevalence of smoking to be 52% in males and 2.7% in females.

If, as seen in blue collar workers, an average of 16 bidis or 11 cigarettes are smoked per day per person (10), the total number of smokers would drop to 107 million in the population and if as above 5% of these are females, then the prevalence of smokers would be 48.4% in males and 2.5% in females. From the above rationale, it is not unreasonable to deduce prevalence of smoking habit to be about 50% in males and 2.5% in females.

Thirty-five million kilograms of chewing tobacco was cleared for domestic consumption in 1981 in the country. Prevalence of chewing higher than 10% seems unrealistic as the average consumption per day per chewer dwindles to negligible amounts. On the basis of 10% prevalence there would be 42 million chewers in the population. Adjustment for sex differential is not attempted as it is not clearly discernible from the available data.

Burden of tobacco related diseases in the country

The number of prevalent/incident cases and deaths of tobacco related diseases, have been estimated from the available data. Furthermore, the proportion of these cases/deaths attributable to tobacco habit is estimated by the formula [p(RR-1) / (p (RR-1) + 1)] where RR is the risk ratio and p is the fraction of the population with habit. For the purposes of the present analysis, estimates of RR, whenever these are documented in Indian studies, covered in the earlier part of the paper, have been utilised. If not, risk estimates available from western studies, when applicable, have been employed. The fraction (p) of the population with tobacco habits is taken as estimated in the preceding section. The number of cases/deaths attributable to tobacco use and hence avoidable are estimated for the 1986 population of 760 million individuals (males : 390 million, females : 370 million).

Morbidity

Major tobacco related cancers

Reliable estimates of incidence of cancer are available from population based registries located at three different cities in the country viz., Bombay, Bangalore and Madras. These have been utilised to arrive at estimates of the number of new cases of cancer of relevant sites, expected to occur in the country (28). Information on risks associated with tobacco habits is available for sites in the upper alimentary and respiratory tracts. Other sites like bladder and pancreas though known to be associated with tobacco use, are not considered because of non-availability of associated risks. Extrapolation of risks from western studies is not justified since the effect that tobacco chewing may have on these sites is unknown.

Thus the estimates of number of new cases of cancers per year for sites in the upper alimentary and respiratory tracts together with the number avoidable, worked out on the basis of habit-specific risks for each sex for the 1986 population, are shown in table 4. It is seen that 61% of oral cancers, 79% of pharyngeal and laryngeal cancers, 34% of oesophageal cancers and 75% of lung cancers i.e. a total of 63% of new cases of cancers at these sites are avoidable. The incidences of bladder and pancreatic cancers are low in the population, together contributing around 19,500 cases. Only a certain fraction of these will be attributable to tobacco use and their inclusion would not make an appreciable difference to the total number arrived at as avoidable.

Table 4. Estimates of incident and avoidable cancers of tobacco related sites[+] in the country (1986 population)

Site (ICD 9th)	Estimated number of cancer cases (in thousands)					
	Total cases			Avoidable cases		
	Male	Female	Total	Male	Female	Total
Oral cavity (140–145)	31	25	56	25	9	34 (61%)
Pharynx and Larynx (146–149, 161)	42	10	52	38	3	41 (79%)
Esophagus (150)	20	15	35	10	2	12 (34%)
Lung (162)	24	4	28	20	1	21 (75%)
Total	117	54	171	93 (79%)	15 (28%)	108 (63%)

[+] Sites like bladder and pancreas are not included for reasons stated in the text.

Proportion avoidable as percent of total are shown in parenthesis

From this incidence data, prevalence of tobacco related cancers in the population, approximated as 2.5-fold of incident cases, is estimated to be 427,500 of which 270,000 are avoidable.

Cardiovascular disease

Incidence of CVD for both sexes in different age groups is not available in the country. From the data of one cohort study (10), incidence of CHD in males in the age group 40 to 59 years, can be estimated to vary between 5 to 7 cases per 1000 per year, giving 320,000 to 448,000 new cases per year, in this age group in the population.

There is very limited data available on prevalence. Prevalence of CHD has been reported in a study from Haryana (N. India), based on ECG taken at rest (29). Since the prevalence for rural areas have been shown to be almost a third or a fourth of urban areas, age-sex specific rural rates have been applied to 80% of the population of the country, semi-urban rates to 10% of the population and urban rates to the remaining 10% of the population. The estimate of prevalence of CHD in the population is computed to be approximately 6 million cases (males : 3.6 million and females : 2.4 million) in the age group 30 and above. Crude estimate of number of cases of cerebrovascular disease is obtained as 433,000 from one study cited by Dalal (30) which reports prevalence of "Hemiplegic" as 5.7/10,000 in a survey conducted in a region near Vellore in South India. Similarly prevalence of peripheral vascular disease is also reported in one M.D. thesis as 2.5% in the age group 40 and above, cited by Sapru (31), giving a crude estimate of 4 million cases in the population.

The risk of smokers to develop CHD, as seen in Table 3, varies from 2 to 3-fold. Taking a conservative estimate of 2-fold risk, the number of cases attributable to smoking works out to be almost 1.3 million, i.e. 21% of prevalent CHD cases. The risks of smokers to develop cerebrovascular disease or peripheral vascular disease are not available and hence estimate of proportion of cases attributable to smoking cannot be arrived at.

Chronic obstructive lung disease

There is no information on COLD incidence and data on prevalence of chronic bronchitis is available from studies carried out mostly in the northwest of the country. These studies give crude rates varying between 0.8% and 12.5% for males and 0.6% to 5.0% for females (32).. Age-sex-specific prevalence rates based on objective tests are available for Chandigarh (N. India). It is reported that chronic bronchitis is much more common in the north than in the south of the country. Hence, instead of extrapolating Chandigarh rates to the whole country, a plausible range of prevalence estimates were obtained by applying these rates to a fraction (varying from 0.50 to 0.25) of the population and to the remaining fraction, half, one-third, and one-fourth the Chandigarh rates. The range of estimates of prevalence of chronic bronchitis thus obtained were between 15 and 29 million (33).

With a 4-fold prevalence of COLD in smokers (23) the proportion of cases attributable to smoking and hence avoidable ranges between 7 million to 13 million.

Other conditions

Association of smoking by the mother during pregnancy and low birth weight of the child has been documented in studies undertaken in the west. Paucity of data, prevents us from addressing this problem, as well as to the effects of passive smoking for the Indian population.

The estimates arrived at in this section, on the prevalence of various tobacco-related conditions together with the number avoidable, have been utilised in the summary table 7 of the final section which gives an overview.

Mortality

Major tobacco related cancers

No reliable data on cancer mortality are available in the country, except for Bombay city, as generated by Bombay Cancer Registry (34). On application of these age-sex-specific mortality rates to the population of the country, the number of deaths due to cancers of oral cavity, pharynx plus larynx, oesophagus and lung were estimated to be 10, 27, 18 and 22 thousand respectively in males and 6, 7, 13 and 5 thousand in females. However, the incidence of these cancers are known to vary depending on the tobacco habits prevalent in different regions of the country. Hence another set of estimates were obtained by applying to the incident cases, the mortality to morbidity ratios observed in Bombay city over a period of 3 years, 1983, 1984 and 1985. These estimates of deaths, shown in Table 5, differ

Table 5. Estimates of cancer deaths and avoidable deaths of tobacco related sites[+] in the country (1986 population)

Site (ICD 9th)	Estimated number of cancer deaths (in thousands)					
	Total deaths			Avoidable deaths		
	Male	Female	Total	Male	Female	Total
Oral cavity (140–145)	11	9	20	9	3	12
Pharynx and Larynx (146–149, 161)	27	7	34	25	2	27
Esophagus (150)	17	13	30	9	2	11
Lung (162)	22	4	26	18	1	19
Total	77	33	110	61 (79%)	8 (24%)	69 (63%)

[+] See footnote table 4.

marginally from the ones mentioned above, except for oral cavity cancer deaths in females, which increased from 6 to 9 thousand.

Cancer mortality risk ratios are not available. Utilization of incident risk ratios of Table 2 would provide an approximation of the number of deaths attributable to tobacco use, and these have been shown in Table 5 as avoidable deaths along with total deaths for each site. It is seen that overall 63% of these deaths are avoidable, the same naturally as for incident cases (the difference in females is due to small numbers and rounding).

Cardiovascular disease

The only available information on deaths due to CVD is provided by the Bombay Municipal Corporation. Applying age-sex specific death rates from this source (35) one obtains an estimate of 700,000 deaths due to CHD and 200,000 deaths due to cerebrovascular disease in those 15 years of age and above in the country. There were only 162 deaths in Bombay city due to "other diseases of arteries" and age-sex-specific information was not available. Utilising the crude proportion (deaths due to this cause to all causes) gave an estimate of 22,000 deaths in the country (Table 6). These estimates may be considered on the higher side, because of urban-rural differences in particular for CHD , but the better health-care infrastructure available in the city would lead to lower mortality, thus to some extent offsetting the effect of the former. The second crude estimate of 450,000 deaths due to CHD can be obtained from the Registrar General's Report (36) which gives 4.6% of all deaths in country (1983) to be due to "heart-attack". This is almost half of Bombay's proportion, which is 11% in males and 8% in females. Similarly if the proportions could be assumed to be halved for the other two conditions, an estimate of 120,000 deaths due to cerebrovascular disease and 11,000 deaths due to "other disease of arteries" would be obtained. A third estimate of CHD deaths (300,000) can be obtained by taking the ratio of mortality to estimated prevalence of the disease in Bombay city and applying it to the

Table 6. Estimates of CVD and COLD deaths in the country (1986 population)

ICD 8th code no.	Cause of death	Range of total deaths (in thousands)	Range of avoidable deaths (in thousands)
410–414	Ischemic heart disease	300–700	66–151 (22%)
430–438	Cerebrovascular disease	120–200	22–35 (18%)
440–448	Other diseases of arteries	11–22	–
490–493	Bronchitis, emphysema, asthma	250–460	110–210 (45%)

Avoidable deaths as percent of total are given in parenthesis.

population (country) prevalence. This would give an underestimate because of better health-care available in the city. Thus a range of estimates for the adult population can be obtained and are shown in Table 6.

Mortality risk ratios of smokers for these conditions, which are needed to estimate the number of attributable deaths, are not available from Indian studies. Surgeon General's Report (2) gives CHD mortality rate ratio (RR) of cigarette smokers to vary from 1.58 to 2-fold for males and 1.30 to 1.78 for females, as compared to nonsmokers. Assuming RR to be 2 for all types of smokers, which is not an unreasonable assumption in view of the fact that bidi smoke has been found to have as deleterious an effect as cigarette smoke on several parameters known to be related to the pathogenesis of this disease (19–21), the proportion of deaths attributable to smoking and hence avoidable are estimated and shown in table 6. It is seen that 22% of CHD deaths are attributable to smoking.

Similarly, assuming cerebrovascular disease risk of smokers to be 2-fold [mortality ratio ranged from 1.3–2.8 for male and 1.2–2.9 for female cigarette smokers (2)], the estimate of the number of avoidable deaths are obtained and shown in table 6 to be 18% of all cerebrovascular deaths.

Chronic obstructive lung disease

Just as for CVD, death records of Bombay city provide the only available data on mortality (35). However, deaths due to chronic bronchitis, emphysema and asthma are clubbed together in reporting cause of death. It is generally accepted that prognosis is good for asthma and mortality is very much lower than for chronic bronchitis or emphysema. Therefore, in the absence of more precise data, mortality rate for the combined group of chronic bronchitis, emphysema and asthma has been considered to represent mortality due to COLD for the purpose of estimating number of deaths in the country.

Applying the age-sex-specific death rates of Bombay city to the whole population gives an estimate of 400,000 deaths in the country due to COLD. Other estimates ranging from 250,000 to 460,000 deaths have been obtained (33) by applying a justifiable ratio of mortality to morbidity (1.6%) to the various estimates of population prevalence given earlier.

The mortality risk ratios for bronchitis and emphysema as reported for the U.S. Veterans (37),were 4.8-fold for light smokers (1–9/day) and 22-fold for heavy smokers (40 + / day). It is not unreasonable to take the risk to be 4-fold, since it is also the risk in smokers for morbidity from COLD. The number of deaths attributable to smoking, using this risk figure, are shown in table 6 as avoidable deaths, and form about 45% of total COLD deaths.

Other conditions

There are a few studies in Indian populations which have documented effect of maternal tobacco chewing and pregnancy outcome. We have addressed ourselves

to the effect of maternal tobacco chewing on stillbirths, since these could be relatively reliably estimated. There were 30 stillbirths per 1000 live births reported for Bombay city, for the year 1981, by its municipal corporation (35). Extrapolating from this data, gives an estimate of 700,000 stillbirths in the country. Utilizing the reported 3-fold risk (24), of stillborn offspring in women who chew tobacco, results in 119,000 stillbirths attributable to tobacco chewing by mothers during pregnancy and hence avoidable on elimination of this habit.

Overview

To obtain an overall perspective, the number of cases/deaths from the above considered tobacco related diseases are pooled together and an estimate of the load of prevalent cases and deaths due to these causes in the adult population, together with the number avoidable if tobacco habit could be eliminated from the population, is ascertained.

Morbidity

It is seen from table 7 that out of 33 million prevalent cases of tobacco related diseases considered, 22 million (midpoint of the range 15–29 million) are accounted for by COLD, another 10 million by cardiovascular disease and about half a million by cancer.

Cancer morbidity information is obtained from well-established registries run in different parts of the country. However, only limited information is available on morbidity of other conditions. It would therefore be of interest to compare our estimates with those obtained by Sapru (31) who had undertaken a similar exercise.

Sapru (31) has estimated (a) the prevalence of CHD as 3.4 million for the 1981 population of the country, assuming the rate to be 2.5% for age group 40 and above. That would give an estimate of 4.25 million cases for the 1986 population. Since we have applied age-sex-specific rates, taking into account urban- rural differences as well, the estimate of 6 million CHD cases in the age group 30 and above is likely to be closer to the true prevalence, which even so may well be an under-estimate because of the use of prevalence rates of 1970s. (b) Peripheral vascular disease prevalence is no different from Sapru's, since it is estimated from the same source. Cerebrovascular disease has not been considered separately by him. (c) The estimate of COLD prevalence has been reported by Sapru (31) to vary between 1.6% and 9.0% but for the purposes of computation he has assumed a prevalence of only 3% for the age group 15 and above. This would yield only 14.1 million cases of COLD for the 1986 population. Application of reported age-sex-specific rates in various plausible combinations discussed earlier has yielded (33) a range of rates, from 3.2% to 6.4%, for prevalence of chronic bronchitis in the country. The number of 22 million prevalent cases obtained at the midpoint of the range (4.7%) and entered in the table for assessment of avoidable cases, is more likely to be a reflection of the true population prevalence.

Table 7. Estimates of prevalent and avoidable cases of specified diseases, in the country (1986 population)

Disease	Total no. of cases (in thousands)			No. of avoidable cases (in thousands)		
	Male	Female	Total	Male	Female	Total
Cancer(a)	293	135	428	233	37	270 (63%)
Cancer(b)	33	16	49	–	–	?
CHD (age ⩾ 30 yrs.)	3,600	2,400	6,000	1,212	58	1,270 (21%)
Cerebrovascular dis	–	–	433	–	–	?
Peripheral vascular dis (age ⩾ 40 yrs.)	–	–	4,000	–	–	?
COLD (age ⩾ 15 yrs.)	15,700	6,300	22,000	9,420	440	9,860 (45%)
Total	19,626	8,851	32,910	10,865 (55%)	535 (6%)	11,400 (35%)

Avoidable cases are percent (shown in parenthesis.) of corresponding total cases.
(a) Cancers of upper alimentary and respiratory tracts.
(b) Other tobacco-related cancers.

The highest proportion of cases attributable to tobacco habit and hence avoidable, is observed (table 7) for cancers of the upper alimentary and respiratory tract (63%). In overall terms though, the impact is limited since these cancers form only 1% of the total morbidity. Next in rank comes COLD with 45% avoidable cases, and then coronary heart disease with 21% avoidable cases. This adds upto a conservative estimate of 35% of these diseases to be avoidable, if tobacco smoking and chewing could be eliminated from the population. The avoidable proportion is much higher in males (55%) than in females (6%).

Mortality

The number of deaths by cause and the number of avoidable deaths, for both sexes as well as the combined group are summarised in table 8. Earlier, we have shown (table 6) a range of estimates of deaths for CVD and COLD. Midpoint or a number slightly lower, has been selected from this range to enter into this summary table.

In India, out of 9.8 million estimated deaths in 1986, a little over 5 million deaths are in the age group 15 and above. It is seen from table 8 that almost a million deaths i.e. 21% of the deaths in adults are estimated to be due to tobacco related diseases viz., cancers of upper alimentary and respiratory tract, cardio-vascular disease and chronic obstructive lung disease.

Table 8. Estimates of total and avoidable deaths by cause in the adult (⩾ 15 yrs.) population of the country (1986)

Cause of death	Total deaths (in thousands)			Avoidable deaths (in thousands)		
	Male	Female	Total	Male	Female	Total
Cancer(a)	77	33	110	61	8	69 (63%)+
CHD	290	160	450	97	4	101 (22%)+
Cerebrovascular disease	75	75	150	25	2	27 (18%)+
COLD	250	100	350	150	7	157 (45%)+
Total	692	368	1,060 (21%)	333 (63%)	21 (21%)	354 (56%)
All causes			5,000 (100%)	530 (100%)	99 (100%)	629 (100%)

(a) Cancers of upper alimentary and respiratory tracts.
+ Avoidable deaths as percent of corresponding total deaths.

Estimates of deaths for the specified causes can be compared with the proportion of cause-specific deaths to all deaths cited by Sapru (31) from a report of Registrar General of India, 1979. (a) Deaths due to heart diseases is given as 5.1% of all deaths. Crude extrapolation gives approximately 500,000 deaths due to heart disease in the country for the year 1986 (all ages). The estimate of 450,000 CHD deaths, in the population age 15 and above obtained by us seems reasonable. (b) Deaths due to "coughs" (excluding tuberculosis) is given as 14.6% of all deaths, yielding almost 1,436,000 deaths in the country for the year 1986. This figure, obviously includes not only COLD deaths but also deaths due to pneumonia. In Bombay, deaths due to COLD form 26.3% of all deaths due to respiratory diseases other than tuberculosis. If a similar situation prevails in the country, then the number of COLD deaths would be 378,000. Hence, the estimate of 350,000 COLD deaths in those 15 years of age and above, again seems reasonable. (c) All cancer deaths have been given in Sapru's paper as forming 2.52% of all deaths. Crude extrapolation gives 247,000 cancer deaths in the country for the year 1986. Using the age-sex-specific death rates from the same source which was utilised for estimating upper- alimentary and respiratory tract cancer deaths, gives a total of 293,500 cancer deaths, which is close to the crude estimate shown above. (d) Information on deaths due to cerebrovascular diseases is not given in Sapru's paper.

This exercise increases the confidence in the estimates of cause-specific deaths, that we have arrived at.

Furthermore, of the 5 million deaths from all causes in the age group 15 and

above, a conservative estimate of 629,000 deaths (i.e. 12.6%) had been arrived at (26), as being avoidable if tobacco habit is eliminated from the population. The listed causes account for a total of 354,000 as avoidable deaths i.e. 56%. The remaining 44% of deaths might be due to (a) causes where tobacco could be playing a contributory role as in tuberculosis and other infectious respiratory diseases. These are known to be major causes of mortality in the country accounting for an estimated 860,000 deaths (tuberculosis: 600,000, pneumonia: 260,000), (b) causes where the etiology is not fully established (e.g. peptic ulcer) or (c) causes which are unknown.

It is pertinent to add that the bulk of the unaccounted tobacco related cases/deaths are in females, and is perhaps due to causes, as yet unidentified in tobacco chewers.

To conclude, the estimates of number of cases/deaths and the proportions of these attributable to tobacco use have been assessed by using the available relevant epidemiological information, and serious errors in risk assessment are unlikely. However, with addition/change in epidemiological data base, these estimates would need to be revised from time to time.

This paper is the outcome of the discussions of the "Working Group" chaired by Dr. L.D. Sanghvi. Other members of the Group were, P.N. Notani, K.Jayant, P.C. Gupta and S. Krishnamurty.

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Tobacco Spells Death for Millions

Keith Ball[1]

Introduction

Tobacco is probably the single greatest cause of noncommunicable disease and is likely to produce a world pandemic unless urgent preventive action is taken. Every year fresh hazards of smoking are demonstrated. New research is helping to define the risks of passive smoking, the limited advantages of low-tar over high-tar cigarettes, and the effects of tobacco on the reproductive system. Death rates from lung cancer are already falling in some western countries, but in China, eastern Europe, and the USSR, and probably in the Indian sub-continent they are fast rising, and there is likely to be a major epidemic in these parts of the world in the early years of the next century, far exceeding any previous outbreak of cancer. At a later date it will spread to other developing countries where cigarette smoking is only now beginning. Smoking-related cardiovascular and chronic obstructive lung diseases will be an additional burden. An expansion of health education is clearly necessary, but government action to curb the activities of tobacco companies must be the first priority, particularly in the developing countries.

Around a million new cases of lung cancer in the world occur each year as well as many other tobacco related cancers including those of the mouth, pharynx, larynx, oesophogus, bladder, pancreas and probably cervix. Oral cancers, due to bidi smoking, tobacco chewing and the use of pan are major problems in many Asian countries. Coronary heart disease (CHD) and chronic obstructive lung disease (COLD) caused by smoking could greatly add to the number of sufferers. Although lung cancer rates are now falling in the United Kingdom and among the younger age groups in the USA, the spread of cigarette smoking in many countries, including China, the USSR, the countries of eastern Europe, and, especially, the developing countries, indicates that there will be a global pandemic of tobacco-related diseases in the early years of the next century unless major steps are now taken to prevent it.

The prolonged incubation period of many tobacco-related diseases has prevented recognitions of the size of the threat. From the time when tobacco use begins in a population 30, 40 or 50 years may elapse before tobacco related deaths reach epidemic proportions. While cancer of the lung and COLD will be the main cigarette-related diseases in many countries, cardiovascular disease is likely to remain the most important in industrialised nations. In order to avoid the misery

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towards which vast numbers of people are heading, governments should now take forthright preventive action.

The toll of tobacco

Global mortality

In several western countries a fifth of all deaths have been attributed to cigarette smoking. In India alone it is estimated that between a half and one million premature deaths occur each year from tobacco use. The total world mortality from tobacco must amount to several millions each year.

Lung cancer

In western countries lung cancer is by far the most important of the tobacco related cancers. A threefold increase in the duration of regular cigarette smoking from 15 to 45 years can raise the annual incidence of lung cancer by a factor of up to 100 (1). The risk of lung cancer at the age of 60 is very much greater in those who start smoking at 15 years than in those who do not do so until after the age of 20. Where teenage smoking is now increasing as in many developing countries an epidemic of lung and other cancer is almost inevitable. In many western countries marked reduction of tar levels in the past 30 years appears to have produced a decline in lung cancer rates, but not in cardiovascular disease. However, in China, the USSR, Eastern European and developing countries, high tar levels persist. In Italy sales of cigarettes increased rapidly between 1920 and 1940 and as a result death rates from lung cancer in men aged 45 to 54 are among the highest in developed countries (2). Since 1968 lung cancer rates in men have more than doubled in Yugoslavia, Poland and in Hungary. In the USSR lung cancer death rates increased by 55% between 1970 and 1980. By contrast the rates for men aged 45 to 54 have fallen since 1968 by 49% in Scotland, 40% in England and Wales and 35% in Northern Ireland, although the United Kingdom still has one of the highest rates in the world. This shows that the falling rates of cigarettes consumption, together with lowering of tar levels can have a major impact.

Lung cancer is overtaking breast cancer as the commonest malignancy of women in parts of the USA and Scotland and its prevalence is increasing more rapidly in women than in men in many western countries. The activities of the tobacco companies to persuade women to smoke are bringing in a grim reward.

Other cancers

In many Asian countries cancers of the mouth, larynx and pharynx are the major tobacco related malignancies. Cigarettes smoking doubles the risk of bladder cancer in men and increases it to a rather lesser extent in women. Most studies show that the increasing prevalence of pancreatic cancer in many countries is due to smoking. Although pipe and cigar smokers have a much lower risk of lung cancer than cigarette smokers, their risk of developing oral, laryngeal or oesophageal cancer is as great as for cigarette smokers. If both tobacco and

alcohol are used, the risk of suffering from one of these cancers becomes even higher.

Cardiovascular diseases

In a study of British doctors, cardiovascular deaths accounted for about a half the excess mortality from cigarette smoking, and most were due to coronary heart disease (3). In men under 45 years of age, at least 80% of coronary deaths can be attributed to smoking. Cigarette smoking significantly increases the risk of sudden death. In a group of British soldiers under 40 years of age who survived a coronary event, 93% smoked an average of 28 cigarettes daily (4). In a controlled British drug trial of over 17,000 patients with mild to moderate hypertension, smokers had a considerably increased incidence of stroke and coronary heart disease compared with nonsmokers; it was concluded that in hypertensive patients smoking cessation was likely to be much more effective than the use of blood pressure lowering drugs (5). A review of 12 studies on the effects of stopping smoking after a myocardial infarction showed that reductions of up to 50% in the prevalence of further attacks occurred; it was considered that stopping smoking could be the single most effective means of intervention currently available in the management of patients with established coronary heart disease. The Doll and Peto study (3) showed that those under 55 who stopped smoking halved their risk of a fatal heart attack within five years compared with those who continued to smoke. The United States Surgeon General stated in 1983 that cigarette smoking was the most important known modifiable risk factor for coronary heart disease, while the American Heart Association considered it to be the most preventable cause of premature death in the USA.

Since health workers can play a vital role in smoking control campaigns the example they set is particularly important.

Other vascular diseases

Peripheral vascular disease and abdominal aneurysm have been found to be even more closely related to cigarette smoking than is coronary heart disease. At least 95% of patients with intermittent claudication or gangrene of the legs are smokers. Stopping smoking is usually the most effective treatment and if achieved early in the course of the disease, can prevent the need for amputation.

Renal artery stenosis, leading to hypertension, is also related to cigarette smoking. A heightened risk of subarachnoid haemorrhage exists in cigarette smokers, especially among women on oral contraceptives. One study showed that women who used oral contraceptives and smoked cigarettes had 22 times the risk of women who did neither (6).

Chronic obstructive lung disease

Cigarette smoking is by far the most important cause of this disease, especially in people exposed to atmospheric pollution or a dusty work environ-

ment. Airway resistance may increase in adolescents who smoke, and by the time they are young adults many already have pathological changes in their small airways. In a study of 405 school children, cigarette smoking was closely associated with cough, sputum, and shortness of breath on exertion; after two years of smoking more than a few cigarettes a day they already showed evidence of airway obstruction (7). It may take 40-50 years of smoking before severe symptoms develop. The rate of decline in the forced expiratory volume in smokers is significantly greater than in either nonsmokers or people who stop smoking.

Reproductive health

Cigarette smoking has a wide range of effects on reproductive health. Women who smoke during pregnancy increase the risk of stillbirth and neonatal mortality, and their children are liable to measurable delay in physical and intellectual development upto the age of 11 years. Indian women who chew tobacco increase their risk of a stillbirth 3-fold. Decreased fertility may occur in women who smoke : in one study, five years after stopping contraception 10.7% of smokers of more than 20 cigarettes a day but only 5.4% of nonsmokers had not had a child.

In another study, smoking was found to increase the prevalence of impotence in men; this was attributed to arteriosclerotic changes in the penile arteries (8). Some studies have shown that smoking can reduce the number and motility of spermatozoa.

Smoking has also been shown to be associated with an earlier menopause. This may be due to reduced levels of oestrogen which could also increase the risk of osteoporosis.

The increase in the number of women who smoke in many countries has led to growing rates of cigarette-related diseases. Women under 50 years of age who continued to smoke heavily after a myocardial infarction had a fivefold greater risk of relapse than nonsmokers.

Passive smoking

It is now accepted that there are risks to nonsmokers of inhaling other people's smoke. The smoke from the burning end of the cigarette contains considerably more toxic substances than the smoke breathed out by the smoker. Children whose parents smoke have more coughs and are more likely to have bronchitis and pneumonia than children of nonsmokers, particularly in the first year of life. These findings have been supported by the discovery that salivary cotinine levels in 569 children aged 11 – 16 years were nearly eight times higher when both parents smoked than when neither did so.

Studies relating passive smoking to the risk of cancer have recently been reviewed by The International Agency for Research on Cancer which accepts that passive smoking produces some risk of lung cancer. It is likely that it accounts for the premature death from lung cancer of several hundreds of nonsmokers in Britain each year. About a third of cases of lung cancer in nonsmokers who live

with smokers and about a quarter of cases in nonsmokers generally, may be attributed to passive smoking. In view of the risk to many workers exposed to cigarette smoke, the complete physical separation of smokers and nonsmokers or the prohibition of smoking in work places is the only practicable preventive measure.

In the USA some 40 States already restrict smoking in public places and workplace smoking policies have increased dramatically in the 1980s.

Developing countries

The risk of tobacco-induced diseases in developing countries is increasing at an alarming rate (11). More than half the men in most Third World countries smoke tobacco and as a result many will die prematurely. In the UK 36% of men smoke compared with about 65% of men in India and 85% in Nepal. For women the corresponding figures are 32%, 25% and 75%. In many developing countries there have been marked increases of manufactured cigarettes consumed in recent years. Between 1970 and 1980 consumption increased by 62.5% in Pakistan, 40% in India, 32% in Kenya but only 4% in North America. In the UK there has been a 27% fall of cigarette consumption in recent years. The use of bidi, chewing tobacco and pan is a major hazard in the Indian sub-continent. More than 100,000 new cases of oral cancer occur each year in South and South East Asia with poor prospects of survival (12). Lung cancer is one of the three commonest cancers in India, Malaysia and Pakistan and is frequent in both blacks and whites in Zimbabwe.

The introduction in western countries of smokeless tobacco in the form of Skoal Bandits or tobacco teabags is a matter of grave concern and in the USA is becoming a frequent habit amongst some schoolchildren. If this development is not halted an epidemic of oral cancer could develop in industrialised countries.

In several developing countries the epidemic of tobacco related diseases is already starting. In the words of the World Health Organisation in 1979 "In the absence of strong and resolute government action we face the serious probability that the smoking epidemic will have affected the developing world within a decade. Smoking diseases will appear in developing countries before communicable diseases and malnutrition have been controlled."

Smoking control

Since health workers can play a vital role in smoking control campaigns, the example they set is particularly important. In New Zealand, the United Kingdom, and the USA, where doctors' smoking rates have fallen steeply, a fall has also occurred in the prevalence of the habit in the general population. Smoking rates in California among physicians fell from 53% in 1950 to about 10% in 1980 (13). But in many countries more than half the doctors smoke, which is bad for their own health and a bad example to other people. In those countries where smoking rates have fallen, it is usually the medical profession that has taken the initiative. Evidence from Australia, Sudan, the United Kingdom, the USA, and the Scandinavian

countries shows that a well-motivated medical profession can influence governments by reporting on the health consequences of smoking.

In order to produce effective plans for smoking control, surveys of smoking habits should be carried out, especially of doctors, other health professionals and children. Not smoking should become the norm in hospitals and health premises.

Tobacco companies are estimated to spend US$2 billion yearly to advertise their products and to counteract the growing public awareness of the dangers of smoking. Since tobacco advertising tends to neutralize health education, especially among young people, all such advertising and sports sponsorship by tobacco companies should be banned. Strong health warnings should appear on all tobacco packages, the wording being changed from time to time. Progressive increases in taxes on cigarettes should be imposed, as this is an effective means of reducing consumption.

Reports on the health consequences of smoking from the Royal College of Physicians in the UK and the Surgeon General in the USA have had powerful effects on their governments and on public opinion. Comparable reports from appropriate medical authorities in developing countries would be likely to have similar results.

Uncompromising opposition by the medical profession and national governments to the vested interests of the multinational tobacco companies is essential if a pandemic of diseases caused by tobacco is to be avoided. Future generations will look back in anger if we fail to take the necessary effective steps.

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Section II

Toxic Chemicals in Tobacco and Tobacco Products

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Agricultural and Technological Experiments to Reduce Toxic Chemicals in Bidi Smoke

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Experimental bidis from tobacco plants with varying genotypes and grown with differing agricultural practices, as well as bidis with varying physical characteristics were tested for their levels of dry TPM, carbon monoxide, nicotine and phenol in smoke. It was observed that well recognized pre-harvest factors affecting tobacco productivity could alter only marginally the levels of these toxic chemicals in bidi smoke. Certain geographical areas, because of their agro-climatic condition and other edaphic factors, are capable of producing relatively less toxic bidi tobacco than other areas. But very few such areas are under bidi tobacco acreage in the country, so that the net effect again is marginal. Among the various physical modifications attempted, replacement of tendu leaf wrapper by cigarette paper reduced the toxic chemicals in bidi smoke considerably but this procedure had its own piquant problems. Perforation of bidis was another modification, which delivered smoke with less toxic chemicals but were difficult to popularise because of loss of smoking pleasure and smoking strength. Bidis with three different filters (20 mg each, of cotton, amber-scented cotton and cigarette filter) also reduced the toxic components in bidi smoke. However, scented cotton filter bidi not only resulted in maximum reduction in smoke toxicants (46.5% to 71.5%), but consumer appeal was also very high.

Introduction

Bidi is an unique smoking product and native of Indian subcontinent. About 20% of Indian population indulge in bidi smoking and 1600 million bidis are hand-rolled every day to meet the demand of vast consumer market (1). Bidis are approximately one half the weight of a standard size cigarette, and tobacco flakes are wrapped in a forest leaf variously knows as *tendu*, *kendu* and *timru* in almost 1 : 1 ratio. Being a product of rural origin, hand-rolled by village artisans spread almost all over India, there is not much of standardization in physical metrology of the product other than the shape. Manufactured bidis vary widely in weight, length, filling density, all of which profoundly affect smoke characteristics (Table 1).

Health risk factors associated with cigarette smoking have been found to be equally applicable, if not more with bidi smoking. Earlier belief was that since the major weight fraction of manufactured bidi was a non-tobacco product and since bidi tobacco was sun-cured unlike cigarette tobacco which is flue - cured, the

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Abbreviations : TPM : Total particulate matter.

Table 1. Toxic constituents in manufactured bidi smoke

Code No.	Length of bidi ± 2 mm	Weight of bidi ± 30 mg	Filler/ wrapper ratio	No. of puff/ bidi	Dry TPM mg/ bidi	Carbon monoxide mg/bidi	Smoke nicotine mg/bidi	Smoke phenol µg/bidi
A	90	850	1.1	27.3	51.8	41.0	4.4	520
B	75	550	0.8	16.2	34.3	26.2	3.2	335
C	80	490	0.6	18.0	38.4	26.7	2.7	328
D	70	490	1.0	12.7	35.4	24.8	3.2	342
E	60	490	1.0	15.4	32.9	20.7	3.5	362
F	60	370	0.9	8.9	23.3	21.2	2.3	215
G	60	250	0.7	10.0	20.8	14.3	2.0	210

associated toxic components in bidi smoke were lower than cigarette smoke. This however has been shown to be erroneous (2). Intensive search, therefore, is in progress to identify pre- harvest and post-harvest factors and product modification procedures which could reduce the harmful effects of toxic components in manufactured bidi and offer partial protection to those who would continue bidi smoking inspite of antismoking campaign.

Of the various toxic parameters in bidi smoke, we have studied, dry TPM, gasphase carbon monoxide, smoke nicotine and smoke phenol of bidi tobacco produced under diverse field backgrounds and product modifications. Implication of these toxic components are well recognised. Dry TPM is considered to be a tumor initiator, gas phase carbon monoxide and smoke nicotine have a role in cardiovascular and pulmonary diseases and smoke phenols are potential tumor promotors.

Estimation procedure for toxic components

All tobaccos were grown in our experimental research farm during 1983-84 and 1984-85 and sun-cured at the growing site. They were brought to the laboratory and the prepared flakes were hand rolled for a popular medium size (60 ± 2 mm) bidi. These bidis were rolled by the same person and tendu leaf was used from a single stock to reduce experimental variability. All the prepared bidis were weight-sorted (490 ± 30 mg) and machine smoked in eight channel automatic smoking machine (Fitrona Model 302) fitted with infrared gas analyser specific for carbon monoxide (ATCOM 352 A) after equilibrating the sample bidis at 60 ± 2% RH and 22 ± 2° C for 72 hours. Bidis in all experiments were tested under the following conditions : Puff interval 30 seconds, puff duration 2 seconds, puff volume 35 ml and butt length 30 ±2 mm in four replications. Tar deposited on cambridge filter pad from four successive bidis was dried in a dessicator over fused calcium chloride for 72 hours to get dry TPM data. Sixteen bidis were smoked per experiment and the average values were computed for dry TPM and

carbon monoxide. Filter pads were exhaustively extracted with methanol and aliquots were used for spectro-photometric estimation of smoke nicotine (3), and smoke phenol (4).

Several types of experimental bidis were prepared and tested for the above mentioned four smoke constituents and will be discussed in the following sections.

Genetic variability

The smoke constituents of bidis made of tobacco of different genetic varieties is presented in Table 2. It may be observed that current cultivars do not differ among themselves with respect to these smoke constituents. Wernsman *et al.* (5) observed that different tobacco classes varied widely in PMI (Particulate matter index). Differences within a class were small but not always consistent under different environmental conditions. Genetic alteration of these constituents in bidi tobacco will probably require introduction of germplasms from diverse sources into varieties or breeding lines. Although a vast number of genetic germplasms in the form of varieties and species is available for breeding programme, the growers' demand for high yielding varieties, crop and product uniformity and high quality leaf, have of necessity, not allowed selection for tobacco varieties which would yield lower toxic constituents when smoked. New perspective of social responsibility for public health issues may now require incisive search for germplasm modification towards 'low risk' tobacco. In this respect new bio-technological techniques like clonal propagation, soma clonal variation and recombinant DNA technique may be of potential importance.

Table 2. Varietal influence on toxic constituents in bidi smoke

Variety			Dry TPM	Carbon	Smoke	Smoke
Name	Yield kg/ha	% area culti-vation	mg/bidi	monoxide mg/bidi	nicotine mg/bidi	phenol µg/bidi
A-2	1882	5	29.2	23.9	3.83	279
GT-4	2475	10	31.9	23.8	3.89	254
A-119	2273	85	31.2	24.1	3.01	239
GT-5	2387	Released in 1985	30.4	24.6	3.80	280

Agricultural practices

Influence on smoke chemistry of various agronomic factors which normally affect crop productivity and its quality, is reported in Table 3. It is interesting to note that none of the seven parameters–transplanting dates, nitrogen levels,

sources of nitrogen, irrigation, plant density, topping levels and harvesting dates–elicited any differential response with respect to these smoke characteristics. Manipulation of field practices, thus, hardly has any scope to reduce the toxic chemicals of bidi smoke. Influence of some of these field practices on conventional leaf quality, through alteration of primary metabolites like carbohydrates and nitrogenous constituents, is well known.

Table 3. Agronomic factors influencing toxic constituents of bidi smoke

Cultural practices	Dry TPM mg/bidi	Carbon monoxide mg/bidi	Smoke nicotine mg/bidi	Smoke phenols µg/bidi
A. Transplanting date				
15th August	32.6	24.6	4.88	267
30th August	31.2	23.7	4.96	267
B. Nitrogen level				
200 kg/hectare	27.4	22.1	3.16	225
300 "	29.1	22.8	3.55	265
400 "	30.0	23.6	3.75	300
500 "	29.1	23.9	3.92	295
600 "	29.9	23.4	3.59	294
C. Source of nitrogen				
Ammonium sulphate	28.8	23.6	3.49	254
Urea	28.7	22.9	3.83	267
Anhydrous ammonia	27.6	22.7	3.37	266
D. Irrigation				
No irrigation	29.9	24.7	3.48	246
As and when required	29.5	23.3	3.71	257
E. Plant density				
18520/hectare	31.8	24.8	4.73	257
22220/hectare	32.1	23.6	5.23	276
F. Topping level				
12-leaves	31.0	24.4	4.40	304
18-leaves	32.2	23.6	3.80	280
G. Harvesting date				
140 DAT *	29.2	22.8	3.60	248
160 "	29.4	22.3	3.90	260
180 "	28.2	21.9	3.60	238

* Days after transplanting.

However, smoke chemistry is also dependent upon secondary metabolites like diterpenes, waxes, phenolics, sterols and alkaloids. Influence of cultural practices on these secondary metabolites except alkaloid has not been clearly understood. Routine agronomic practices thus have limited scope in reducing health hazardous components of bidi smoke. One can, however, conjecture that spraying field crop with sublethal dose of certain chemicals like metal chelating compounds (EDTA-Sodium Salt) or bacterio-static chemicals (e.g. sulphamic acid, sodium salt of penicillin) or enzyme co-factor additive (zinc and other micronutrients) may affect the growing leaf metabolism in an unknown way which might result in altering the levels of carcinogenic and co-carcinogenic compounds in tobacco leaf. Producing tobacco in CO_2 enriched atmosphere have resulted in increased leaf yield by 7% and decreased the accumulation of protein, nitrate, alkaloid and nitrogen (6), which are well known leaf precursors of toxic chemicals including nitrosamines in tobacco smoke. Increasing CO_2 concentration in the crop canopy can be attained by spraying carbonated water on leaves and/or resorting to practice of organic manures as fertiliser. Such methods are worth considering in the context of reducing toxic chemicals in bidi smoke. Work is being persued in these promising areas.

Environmental factors

Table 4 shows the effect of environmental factors on bidi smoke constituents. Environment affects the developmental physiology of a growing tobacco plant in a profound way and influences cured leaf quality (7). This results in altered smoke chemistry. The levels of toxic chemicals particularly dry TPM and smoke nicotine

Table 4. Environmental factors affecting smoke constituents in bidi tobacco

	Dry TPM mg/bidi	Carbon monoxide mg/bidi	Smoke nicotine mg/bidi	Smoke phenol µg/bidi
A. Season				
1983-84	28.7	23.2	3.85	257
1984-85	33.8	24.9	4.94	281
% Reduction in 1983-84 over 1984-85	15.1	6.8	22.1	8.5
B. Location (1983-84)				
Anand	26.1	25.3	3.30	253
Nipani	18.2	20.0	1.80	209
% Reduction in Nipani over Anand	30.3	20.9	45.5	17.4

are seen (Table 4) to be reduced by 15% and 22% respectively in smoke of bidi tobacco grown in 1983-84 as compared to next year's crop. Locational differences were even more marked. Bidis made of Nipani tobacco were characteristically less toxic with respect to all four categories of chemicals than bidis made of tobacco grown at Anand. Dry TPM was reduced by almost one-third, smoke nicotine by one-half, carbon monoxide by one-fifth and smoke phenol by one-sixth in bidis made from Nipani tobacco than those from Anand.

Seasonal features of these two crop-growing years are presented in Table 5. Although it is difficult to isolate factors like diurnal fluctuation of temperature and day to day fluctuation as the season progresses with the interacting effects of relative humidity, rainfall and sunshine hours in field crop, tobacco growing season of 1983-84 (17th Sept. to 31st Dec.) was characterized by widely distributed high rainfall with less diurnal variation of temperature as compared to the following season. Such climatic conditions have been reported (8,9) to be conducive for producing lower nicotine content in tobacco leaves. This is reflected in the smoke nicotine data (Table 4). The levels of nicotine in bidis of 1983-84 tobacco produce are seen to be lower by 22% over the 1984-85 produce. There is a paucity of information on the environmental effect on TPM content. However, many workers have observed direct correlation of smoke nicotine with tar delivery (10,11). Relatively low TPM levels in the smoke of bidis of 1983-84 season is in conformity with these observations.

Locational characteristics of Anand and Nipani are presented in Table 6. Here again, climatologically contrasting situation similar to the one described above is in operation. Very widely distributed rainfall with much less diurnal variation in atmospheric temperature (which is on the cooler side because of the high altitude)

Table 5. Seasonal features

	Transplanting (20th Aug.– 16th Sept.)	Growth (17th Sept.– 31st Dec.)	Maturation (1st Jan.– 1st April)
1983–84			
Rainy days (No.)	10	6	—
Rainfall (mm)	118.6	77.8	—
Max. temp. (°C)	32.88	32.14	32.64
Min. temp. (°C)	25.35	16.65	12.45
Diurnal diff. (°C)	7.53	15.49	20.19
1984–85			
Rainy days (No.)	8	3	—
Rainfall (mm)	61.1	41.9	—
Max. temp (°C)	30.71	33.47	34.87
Min. temp. (°C)	23.64	16.43	13.53
Diurnal diff. (°C)	7.07	16.84	21.34

prevails at Nipani during the crop growing season than at Anand. Further, Nipani soil may favour better nutrient mobilization (both macro and micro) because of its more neutral pH condition and use of balanced fertilizer (nitrogen- phosphorous and potassium). Moreover, being a rainfed crop, unlike Anand tobacco crop which is irrigated, there should not be uneven flux in nutrient uptake, mobilization and metabolic conversion. All these factors may affect chemical composition of the leaf, particularly its secondary metabolites, giving rise to less toxic constituents in bidis made from tobacco grown at Nipani.

Table 6. Locational features

	Anand	Nipani
Soil type	Loamy sand	Silt loam
Soil pH	8.1	7.1
Altitude (MSL, M)	45.8	610.0
Rainy days (No.)	16.0	43.0
Rainfall (mm)	196.4	200.2
Maximum temp. (°C)	32.55	26.8
Minimum temp. (°C)	18.15	21.7
Diurnal diff. (°C)	14.40	5.1
Fertilization (kg/hectare)	18ON : OP_2O5 : OK_2O	14ON : $4OP_2O5$: $2OK_2O$

Processing practices

Bidi makers use various flake sizes of filler tobacco depending upon prevalent practices of the area and brand names. They also use fermented tobacco of 'some sorts' depending upon consumers' response without any rigorous process control.

Bidis were made in our laboratory using same supply of tobacco filler of various flake sizes (6 to 14 nos.) and tested for influence of flake size on toxic chemicals in bidi smoke. Results are presented in Table 7. Similarly, tobacco fillers were heavily 'ordered' (22 ± 2% moisture content) and fermented at 50 ± 2°C and 75 ± 5% RH for a variable period ranging from 3 days to 15 days with different additives i.e. a) water to reactivate residual enzyme in dry tobacco (normally fermented); b) baker's yeast suspension as an extraneous source materials for oxido-reductase group of enzymes as in glycolytic fermentation; c) tobacco seed wash suspension as a source material for extracellular bacterial enzymes and d) tobacco root wash as a rich source material for peroxidase group of enzymes. After stipulated period of fermentation, tobaccos were 'reordered' and bidis were made out of these fermented tobaccos and tested for toxic chemicals in smoke. These results are also presented in Table 7.

Table 7. Processing factors affecting bidi smoke constituents

	Dry TPM mg/bidi	Carbon monoxide mg/bidi	Smoke nicotine mg/bidi	Smoke phenol µg/bidi
A. Flake size (1983-84 produce)				
6 No.	22.0	22.8	2.51	228
8 No.	24.2	22.8	2.82	248
10 No.	20.6	21.1	2.38	216
12 No.	22.5	23.1	2.68	242
14 No.	21.4	23.4	2.51	221
B. Fermentation (1985-86 produce)				
Plain tobacco (Control)	36.1	25.6	3.63	225
Normally fermented	36.7	25.3	4.74	205
Yeast fermented	37.2	27.1	3.73	198
Seedwash fermented	37.6	27.3	4.65	225
Rootwash fermented	36.5	25.6	4.34	210

It is seen from the data that neither the flake size nor the fermentation process had any effect on reducing toxic chemicals in bidi smoke.

Product modification

A breakthrough in reducing the toxic chemicals in bidi smoke was obtained only by product modifications. Table 8 presents the data obtained in modified bidi i.e. bidi tobacco wrapped in thin cigarette paper, and compares it with regular bidi which has a tendu leaf wrapper. About 60% reduction in dry TPM and smoke nicotine, 70% reduction in carbon monoxide and 90% reduction in smoke phenol was achieved by this modification. Experimental data thus show that the toxicity of bidi smoke can be very substantially reduced if only wrapper is changed from the usual tendu leaf to thin paper. However, this modification has its own practical difficulties. For one thing it calls for a drastic policy change affecting tendu leaf industry and its export market which by itself is a multimillion rupee trade in India. For another, rolling hard bidi flakes in delicate tissue paper easily pierces the wrapper. To avoid such difficulties either the textural change in bidi flakes or change over to machine making of bidi or both are essential. This also calls for a significant change in the organisational set up which may be difficult in a traditional society resistant to change.

A relatively simpler way of reducing toxic chemicals in bidi smoke, however, is to make a pair of pin holes approximately 20 mm from the smoking end. This will have the ventilation effect of the so called perforated cigarette paper. Experimental

Table 8. Effect of wrapper on toxic constituents in bidi smoke

	Wrapper		% Reduction
	Tendu	Paper	
Wt. range, mg/bidi	340 ± 30	580 ± 30	—
Burnt quantity, mg/bidi			
(a) Tobacco	120	292	—
(b) Wrapper	120	34	—
Dry TPM			
(a) mg/bidi	19.3	17.9	7.3
(b) mg/g, tobacco	160.8	61.3	61.9
Carbon monoxide			
(a) mg/bidi	17.1	13.0	24.0
(b) mg/g, tobacco	142.5	44.5	68.8
Smoke nicotine			
(a) mg/bidi	1.2	1.2	0.0
(b) mg/g, tobacco	10.0	4.1	58.9
Smoke phenol			
(a) μg/bidi	263	58	77.9
(b) μg/g, tobacco	2191.7	198.6	90.9

data obtained with such modified bidis are shown in Table 9. This simple technique which would not require any change in the bidi manufacturing process is potentially capable of reducing dry TPM by 33.8% carbon monoxide by 41.8% smoke nicotine and smoke phenol by 52.1% and 18.8% respectively. The only drawback of this product is that because of air-dilution of bidi puff through pin-

Table 9. Effect of wrapper perforation (2 pin holes) on toxic chemicals in bidi smoke

	Length of bidi ± 2mm	Weight of Bidi ± 30 mg	Dry TPM mg/bidi	Carbon monoxide mg/bidi	Smoke nicotine mg/bidi	Smoke phenol μg/bidi
Normal	60	430	23.1	17.0	2.40	128
Perforated	60	430	15.3	9.9	1.15	104
% Reduction over normal	—	—	33.8	41.8	52.1	18.8

hole ventilation, smoking strength will be proportionately reduced and draw resistance would increase, correspondingly reducing smoking pleasure. Hence consumer appeal to such modified product may be rather poor.

Perhaps a realistic approach to the problem of reducing toxic chemicals in bidi smoke would be introduction of filters in bidi. Various types of filter material could be conceived for such purpose. Data collected on three different filter types are presented in Table 10. Success of various degrees was obtained with different filter materials, each of which weighed 20 mg. Synthetic filter materials used in mechanised cigarette industry neither proved very efficient nor was it easy to incorporate in hand-rolled bidis. Cotton filter was more efficient than cigarette filter in reducing toxic components of smoke and it was rather easy to introduce in hand-rolled bidis without requiring any additional expertise or lowering of production rate. Scented cotton was another filter material tried. This was prepared by soaking cotton in 0.1% alcoholic solution of amber (a natural resin) and then air dried. This treatment not only had flavourful smoke delivery, but because of the fixative nature of the encapsulated resin, enhanced the retention of smoke particulate matter in treated cotton, thus increasing the filtration efficiency. In fact, scented cotton filter reduced dry TPM by 70%, nicotine and phenols by 60% and carbon monoxide by 46% compared to non-filter bidis.

Table 10. Effect of filters on toxic chemicals in bidi smoke

Sr. No.	Filter	Dry TPM mg/bidi	Carbon monoxide mg/bidi	Smoke nicotine mg/bidi	Smoke phenol μg/bidi
1	Non-filter (normal)	29.5	21.5	4.35	251
2	Cigarette filter (20 mg)	18.1	14.7	1.97	184
3	Cotton filter (20 mg)	12.8	15.2	1.59	120
4	Scented cotton filter (20 mg)	8.4	11.5	1.68	100
5	% Reduction in treatment no. 4 over no. 1	71.5	46.5	61.4	60.1

Disadvantages accruing from loss of smoking strength however, were adequately compensated for by rich flavourful smoke; so that overall, consumer appeal for scented cotton filter bidi remained high. Opinion survey of 87 smokers conducted in one sitting at an Agricultural Fair during 1985 indicated (Table 11) that overwhelming majority of the smokers preferred this simple modification in manufactured bidi which very substantially reduced toxic chemicals in bidi smoke. Scented cotton filter bidi thus offers an unique opportunity of combining otherwise incompatible issues : rustic smoking pleasure with low delivery of toxic substances and reducing health risk.

Table 11. Opinion survey on smoking quality of scented filter bidi

Smoking quality parameters	Assessment	Percent of smokers *
Smoking strength :	Mild	11
	Medium	67
	Strong	22
Draw :	Normal	78
	Hard	22
Aroma :	Pleasant	87
	Unpleasant	13
Overall acceptance :	Preferred	90
	Not preferred	10

* A total of 87 smokers interviewed.

On summing up, investigations into various agricultural operations involving tobacco production show that well recognised pre-harvest factors affecting tobacco productivity can hardly reduce the toxic chemicals in bidi smoke except some marginal seasonal effect on which there is no human control. Certain geographical areas, however, because of their agroclimatic conditions and other edaphic factors are capable of producing relatively less toxic bidi tobacco than other areas. Very few such areas are under bidi tobacco acerage in the country and the effect again would be marginal. Change over to paper wrapper may effect substantial reduction in toxic chemicals in bidi smoke but this has its own piquant problems. Perforated bidis deliver less toxic chemicals but they are difficult to popularize because of loss of smoking pleasure and smoking strength. Filter bidis can very significantly reduce the toxic components in bidi smoke. In particular scented cotton filter bidi not only reduces the smoke toxicants varying from 46.5% to 71.5%, but consumer appeal for such modified product has also been shown to be very high.

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Toxic Constituents in Bidi Smoke

K. Jayant[1] and S.S. Pakhale[1]

Bidi, a popular form of smoke in India is smoked by over 130 million persons. Epidemiologic studies have established bidi smoking to be a risk factor in cancers of the upper alimentary and respiratory tract, coronary heart disease and chronic obstructive lung disease.

Levels of harmful constituents in a single bidi smoke were estimated under the internationally recommended smoking conditions for cigarettes except for puff frequency. The bidi had to be smoked at 2 puffs per minute (instead of one) to keep it burning. Chemical analysis of the bidi smoke shows that overall toxicity as measured by total particulate matter (TPM) and nicotine is high (tar 23-41 mg/bidi, nicotine 1.74-2.78 mg/bidi). Hydrogen cyanide, which is the strongest cilia toxic agent in tobacco smoke is estimated to vary from 688 μg to 904 μg per bidi. Phenols are known tumor promoting agents and their levels range from 129 μg to 273 μg per bidi.

Furthermore benzo(a)pyrene which is often used as an indicator of concentration of tumorigenic PAH in the smoke varies from 108 ng to 144 ng. Emission levels of tar and nicotine based on actual smoking behaviour of the bidi smoker, reveal that the bidi smoker is generally exposed to greater health hazard than indicated by standard machine estimates.

Bidis and cigarettes were smoked on the smoking machine (a) as per standard smoking condition for bidis (for a comparative study of product characteristics) and (b) as per smoking behaviour of the respective type of smokers (to assess exposure levels). The long bidi, despite having less amount of tobacco (1/3 that in a cigarette) was found to yield tar and nicotine values similar to filter king cigarettes in the first comparison and even higher than filter king in the second. For the regular bidi, levels of tar were lower than in cigarettes in both the comparisons. However, the nicotine levels were similar to nonfilter and filter (70 mm) cigarettes in the first comparison and similar to filter king in the second.

No low tar–low nicotine bidis are as yet marketed in the country. Various methods of reduction standardised in the laboratory, some of which are suitable for commercial adoption are discussed.

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Abbreviations : TPM : Total particulate matter, HCN : Hydrogen cyanide, B(a)P : Benzo(a)pyrene, CO : Carbon monoxide, PAH : Polycyclic aromatic hydrocarbons.

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Introduction

When tobacco was first discovered in the new world in the early 15th century, it was smoked from a tube or a pipe (1). This form of smoking was followed by cigars and primitive cigarettes obtained by stuffing tobacco in a hollow reed/cane tube or by rolling crushed tobacco leaves in corn husk/other vegetable wrapper. With the first cigarette machine factory which was set up in Havana in Cuba in 1853 (2), cigarette manufacturing spread rapidly to London and the American Colonies. After the first World War, cigarette smoking not only replaced almost all forms of tobacco use but its consumption increased phenomenally (3).

There was concern about the health hazards due to tobacco use ever since it was introduced to the western world. However, systematic scientific studies were initiated only after 1930, when a marked increase in mortality and incidence of diseases like lung cancer were observed. The Report of the College of Physicians of London (4) and the first Report of the US Surgeon General on Smoking and Health in the early sixties (3) evaluated all available epidemiologic and experimental data and came to the conclusion that smoking was undoubtedly causally associated with premature mortality, cancers at certain sites, coronary heart and chronic obstructive lung diseases.

Tobacco was introduced in India in the early 16th Century by the Portugese (5). The Royal Physician in the court of Akbar was apprehensive of the ill effects of smoking and suggested that it be smoked only after passing the smoke through water. This gave rise to the hukkah, which became the prevalent form of smoking in the country. Hukkah is cumbersome to smoke and a convenient form of smoking viz., "bidis" became popular when its manufacture spread from Bihar in 1905 to other parts of the country (6). The hazards of bidi smoking became evident three decades ago, when association of bidi smoking with cancers of the upper alimentary tract was established (7). But it was only in 1974 that the bidi smoke was chemically analysed (8). In recent years, there are not only epidemiologic studies showing bidi smoking to be a risk factor in coronary heart (9) and chronic obstructive lung diseases (10,11) but systematic chemical analysis of bidi smoke of various brands of bidis marketed in the country have also been undertaken (12,13).

Bidi

Bidis are available in 2 lengths, regular (60 mm) and long (80 mm) and are made up of 0.2 to 0.3 g of tobacco which is sun-dried, flaked and rolled in a dried leaf of tendu (Diospyros melanoxylon) or temburni (Diospyros ebenum).

Emission levels of harmful constituents in bidi smoke

The yield of harmful constituents from any smoking stick depends not only on physical and chemical properties like quality and cut of tobacco, curing process, length of stick and quality of wrapper but also on smoking parameters like puff volume, puff duration, puff frequency and butt length. Therefore, for a comparative study of product characteristics, it is necessary to adopt uniform smoking

parameters.

Standard for machine estimates: Federal Trade Commission has laid down the following international standards with regard to cigarettes (14).

1. Conditioning the product in humidity chamber for a minimum of 24 hours at 22 ± 2°C and 60 ± 3% relative humidity.
2. Regulating puff volume at 35 ± 0.5 ml
3. Regulating puff duration at 2 ± 0.2 seconds
4. Regulating puff frequency at 1 puff per minute.
5. Smoking upto butt length of 23 mm for nonfilter cigarettes and 23 mm or length of filter overwrap plus 3 mm whichever is longer, for filter cigarettes.

In adopting the above conditions for estimation of constituents of bidi smoke, it was found that the puff frequency of 1 puff per minute was not adequate to keep the bidi burning. Hoffmann and others (8) who were the first to chemically analyse the bidi smoke, modified the recommended condition of puff frequency of 1 puff per minute to 2 puffs per minute while keeping the other recommended smoking parameters the same as for cigarettes. For a comparative study of standard machine estimates in various brands of bidis the international standard as modified for bidis by Hoffmann et al (8) is followed.

Smoking leads to formation of main stream smoke which is what the smoker is exposed to directly and the side stream smoke which is the smoke in the ambient air between puffs. The estimates given in this paper are restricted to main stream smoke.

Harmful constituents in the tobacco smoke: In the cigarette smoke over 3800 constituents have been identified (15). Although the bidi smoke has not been analysed in such detail, there is no reason to think that the number would be vastly different as both are pyrolised products of tobacco. However, there might be variation in the amounts of the constituents, due to differences in product characteristics.

The smoke condensate from the main stream smoke consists of dry total particulate matter (TPM) and nicotine which are toxic, as most of the carcinogenic and mutagenic agents (so far studied) are known to reside in the particulate phase. Furthermore, nicotine has been shown to act on several physiological responses resulting in increased heart rate and other changes in the normal functioning of the circulatory system (16).

Some of the other specific components that are identified in the smoke and are known to be hazardous to health are hydrogen cyanide, steam volatile phenols, benzo(a)pyrene, and carbon monoxide. Hydrogen cyanide is known to be the strongest cilia toxic agent in tobacco smoke. Its interference with mucociliary clearance which is essential for maintenance of normal pulmonary environment

could result in an accumulation of toxic and tumorigenic agents leading to respiratory diseases. Phenolic fraction of cigarette smoke condensate has been reported to have tumor promoting and carcinogenic activity on mouse skin as also toxic effect on ciliated epithelium. Polynuclear aromatic hydrocarbons in tobacco smoke play an important role in tobacco carcinogenesis and their concentration is often measured by the yield of benzo(a)pyrene. Carbon monoxide, is also present in the smoke and is harmful mainly due to its interference with tissue oxygenation (16).

Table 1. Mean levels of harmful constituents in bidi smoke (under modified international standard conditions)[+]

Type (length) and coded brand names of bidis	Dry TPM (mg/bidi)	Nicotine (mg/bidi)	HCN (μg/bidi)	Phenol (μg/bidi)	B(a)P (ng/bidi)	CO[++] (Vol.%/bidi)
Regular (60 mm)						
BA	25.9 ± 0.50	1.94 ± 0.05	688 ± 37.5	129 ± 12.0	108 ± 10.1	
BC	23.0 ± 0.54	1.74 ± 0.10				
BD	30.0 ± 0.69	2.05 ± 0.08				
Long (80 mm)						
BB	40.8 ± 1.00	2.78 ± 0.13	904 ± 55.9	273 ± 16.4	144 ± 18.8	7.7
BE	38.0 ± 0.68	2.44 ± 0.06				

[+] Puff frequency : 2 puffs per minute, brands marketed in 1979-80.
[++] Brand marketed in 1974 (8).

Levels of harmful constituents in bidi smoke: Levels of the various harmful constituents (described above) are estimated in a single bidi smoke under modified standard conditions and are shown in Table 1. Levels of tar varied from 23.0 to 40.8 mg, nicotine from 1.74 to 2.78 mg, hydrogen cyanide from 688 μg to 904 μg, phenols from 129 μg to 273 μg, B(a)P from 108 ng to 144 ng (12, 13) and carbon monoxide was 7.7 vol % in a long bidi (8).

Comparison of emission levels in bidi and cigarette smoke

Harmful effects of bidi smoke could be better appreciated by comparing the levels of noxious agents in the bidi with those of cigarettes. Locally 3 types of cigarettes are marketed – nonfilter cigarette of 70 mm length and filter cigarettes of 70 mm length and of 85 mm length designated as filter king. Bidis differ from cigarettes in several ways. The amount of tobacco in the bidi is much less (about a fifth to a third) compared to a cigarette, and is sun-cured and flaked as against flu-cured and finely powdered in the cigarette . Furthermore, the bidi wrapper has very poor porosity compared to the cigarette wrapper. These differences lead to

differing emission levels of the harmful constituents in bidi and cigarette smoke.

Table 2. Mean levels of harmful constituents in cigarette smoke under standard smoking conditions modified for bidi[+]

Type (length) and coded brand names of cigarettes	Dry TPM (mg/cig)	Nicotine (mg/cig)
Nonfilter (70 mm)		
CA	36.5 ± 0.71	1.92 ± 0.03
CB	27.4 ± 0.35	1.55 ± 0.01
CC	32.6 ± 0.40	1.72 ± 0.07
Filter (70 mm)		
CD	34.8 ± 0.30	1.92 ± 0.00
CE	32.0 ± 0.15	1.98 ± 0.07
CK	27.8 ± 0.68	1.44 ± 0.06
Filter kings (85 mm)		
CF	37.4 ± 0.48	1.88 ± 0.12
CH	37.8 ± 0.31	1.85 ± 0.01
CJ	41.7 ± 0.94	2.68 ± 0.00

[+] Puff frequency of 2 puffs/min and 23 mm butt length. These conditions also conform to the smoking behaviour of the cigarette smoker.

Comparison of standard machine estimates of levels of emission : Under standard international smoking conditions, the local cigarettes were found to have 18 to 28 mg of tar, 0.9 to 1.8 mg of nicotine. 366 μg to 638 μg of hydrogen cyanide, 118 to 226 μg of phenols and 85 to 114 ng of benzo(a)pyrene (12, 13). As mentioned earlier, for standard estimates, cigarettes are smoked with 1 puff per minute and bidis with two, whereas for product evaluation, both bidis and cigarettes would have to be smoked under similar smoking conditions. When cigarettes were smoked with puff frequency of 2 puffs per minute (like bidis) the tar and nicotine levels varied from 27.4 to 41.7 mg and 1.44 to 2.68 mg respectively, (Table 2). Comparison of these values with those of bidis as given in Table 1 shows that the long bidi despite having only a third of the amount of tobacco in a cigarette has higher levels of tar (38.0–40.8 mg) and nicotine (2.4–2.8 mg) than nonfilter or filter cigarettes (70 mm) but is similar to filter kings (Tar 37.4–41.7 mg, nicotine 1.8–2.7 mg). On the other hand, regular bidi which has about one-fifth of the tobacco in a cigarette, has lower levels of tar (23–30 mg) compared to

cigarettes of all types, but the nicotine yield (1.7–2.0 mg) is generally similar to those of nonfilter and filter (70 mm) cigarettes (1.4–2.0 mg). Levels of other constituents are available for cigarettes when smoked as per international standard conditions (as seen above) but not when smoked at a puff frequency of 2 puffs per minute. However, as tar and nicotine levels could be considered as indicators of overall toxicity, it can be said, in summary, that long bidis are similar to filter kings but are more noxious than nonfilter and filter cigarettes (70 mm), whereas regular bidis are similar to filter and nonfilter cigarettes in nicotine yield but have lower levels of tar compared to all cigarettes.

It needs to be emphasized that comparison of standard machine estimates would only reflect the differences in product characteristics and for a realistic assessment of health hazards due to exposure to one bidi smoke compared to exposure to one cigarette smoke, it is necessary to determine emission levels based on actual smoking behaviour of bidi and cigarette smokers.

Emission levels in bidi and cigarette based on the smoking behaviour of the smoker : To ascertain the smoking behaviour of the smokers, puff frequency and puff duration were observed in about 100 bidi and 100 cigarette smokers and the butt lengths discarded were also measured. It was found that the difference was mainly in puff frequency. Bidi smokers smoked with almost 5 puffs per minute compared to the standard of 2 and cigarette smokers smoked with 2 puffs per minute instead of one. Furthermore, the bidi smoker threw away a longer butt (30 mm) as against the cigarette smoker who discarded a 23 mm butt (12).

Table 3. Mean emission levels in selected brands of bidis based on smoking behaviour of the smokers[+]

Type (length) and coded brand names of bidi		Dry TPM (mg/bidi)	Nicotine (mg/bidi)
Regular (60 mm)	BA	23.20 ± 0.82	2.15 ± 0.07
	BD	25.49 ± 0.74	2.50 ± 0.10
Long (80 mm)	BB	46.47 ± 0.83	3.71 ± 0.09
	BE	46.40 ± 1.08	3.34 ± 0.28

[+] Puff frequency of 5 puffs/min and 30 mm butt length.

The estimates of tar and nicotine levels, in selected brands of bidis, which were smoked on the smoking machine with puff frequency and butt length as

observed in bidi smokers, are shown in Table 3 (13). The smoking behaviour of the cigarette smoker conforms to the standard smoking condition as modified for the bidi. Thus the exposure levels for cigarette smokers are the same as emission levels given in Table 2. The level of tar in regular bidis (23.2–25.5 mg) was lower than for cigarettes (32.0–41.7 mg) but nicotine level in these bidis (2.2–2.5 mg) was higher than in nonfilter and filter cigarettes (70 mm) but was almost similar to filter kings (1.9–2.7 mg). On the other hand, emission levels of both tar and nicotine were much higher for long bidis (tar 46.5 mg, nicotine 3.3–3.7 mg) compared to cigarettes (tar 32.0–41.7 mg, nicotine 1.9–2.7 mg). Thus, exposure due to smoking a long bidi is much higher than due to smoking a cigarette. However, in the case of regular bidi there is as much or greater exposure to nicotine compared to a cigarette although not to tar.

Again, these levels are indirect measures of exposure. Actual exposure, which also depends on inhalation and other smoking parameters would have to be determined from estimation of relevant biochemical and biologic parameters. Study of effect of bidi and cigarette smoking on specific biologic parameters related to pathogenesis of coronary heart and chronic obstructive lung diseases have shown that smoking one bidi is as harmful as smoking one cigarette (17–20).

Reduction of levels of 'tar and nicotine in bidis

As bidi smoking is proven to be a health hazard, every effort should be made to eliminate or reduce the risk of bidi smoking. Undoubtedly, the best approach to reduction of hazards due to smoking is to institute smoking control programmes. No bidi (or cigarette) can be considered safe, however low the emission levels. Even so there are studies (21) which have demonstrated reduction of risk of cancer of the larynx and lung and diseases of coronary artery (22) in long term users of filter cigarettes. Consequently, one of the measures for control of health hazards due to smoking adopted in several western countries is banning of high tar and nicotine cigarettes.

As we have seen, all the bidis marketed in the country have high levels of TPM (tar) and nicotine. As bidi is a product of the cottage industry, there is a need to introduce simple and inexpensive methods of reduction in its manufacture. Some of the methods standardised in the laboratory along with the percentage reduction in each of the modified bidis are shown in Table 4 (23–25).

The first method tried was introduction of filters. Initially rolling of bidis similar to the market bidis was standardised. Then the filter was inserted in the standard bidi. Various filters viz., cotton sliver of 5 mm length and 10 mg weight ($CF_{5,10}$) and 5 mm length 20 mg weight ($CF_{5,20}$) were used. Both the standard and the filter bidis were smoked on the smoking machine as per the smoking behaviour of the smoker. The observed reduction varied from 48% to 68% for tar and 42% to 65% for nicotine (23).

The second method tried was 5 perforations at the 30 mm butt mark of the bidi. Control bidis were similar to market bidis except for perforation. This

Table 4. Mean levels of tar and nicotine and percentage reduction in modified bidis.

Methods of reduction	Tar (mg/bidi)	Nicotine (mg/bidi)	Percentage reduction	
			Tar	Nicotine
1. Introduction of filter				
Control (nonfilter)	23.1 ± 0.64	1.54 ± 0.06		
Cotton filter $(CF_{5,10})^{+}$	12.1 ± 0.51	0.89 ± 0.05	48	42
Cotton filter $(CF_{5,20})^{++}$	7.4 ± 0.31	0.55 ± 0.04	68	65
2. Perforation				
Control (non-perforated)	26.9 ± 0.75	1.93 ± 0.04		
5 perforations	12.9 ± 1.63	0.83 ± 0.01	52	57
3. Substitution of filler tobacco				
Control (usual bidi tobacco)	52.8 ± 0.88	3.90 ± 0.06		
Cigarette tobacco filler	42.7 ± 2.67	1.43 ± 0.04	19	63
4. Substitution of wrapper control (usual tendu-wrapper	23.1 ± 0.64	1.54 ± 0.06		
Cigarette paper wrapper	7.9 ± 0.98	0.69 ± 0.02	66	55

$^{+}CF_{5,10}$ = Cotton filter 5mm of 10mg weight,
$^{++}CF_{5,20}$ = Cotton filter 5mm of 20mg weight.

modification reduced the tar and nicotine by over 50%. It has also been shown that perforation reduces carbon monoxide level (25). As perforation increases draw resistance, optimum number of perforations need to be worked out.

The other two methods attempted were substitution of (a) filler tobacco and (b) wrapper (24). Replacing the bidi filler tobacco by equal amount of processed cigarette tobacco resulted in a 63% reduction in nicotine yield (due perhaps to the superior quality of tobacco filling in the cigarette). But the reduction in tar yield was only 19% which indicates that better quality tobacco is of limited advantage, if the wrapper used has poor combustibility.

In the next experiment, when the less porous tendu leaf was substituted by cigarette paper, there was an appreciable reduction in tar (66%) and nicotine (55%) which again underscores the importance of the wrapper in emission levels.

It is pertinent to mention here, an experiment conducted by Gupta et al (26) in which two groups of subjects were made to smoke equal amounts of bidi tobacco wrapped in bidi and cigarette paper, respectively. They report that in these two groups there was no significant difference in the effect on platelet aggregation, levels of serum free fatty acids and carboxyhemoglobin. This finding which implies

that the two wrappers do not have a differential effect on biological parameters related to coronary heart disease, needs to be confirmed as it is at variance with what might be expected on the basis of our experiment described above.

Conclusion

In conclusion, it can be said that inspite of the small amount of tobacco used in the bidi, emission levels of the harmful constituents are high. Furthermore, the way the bidi is smoked, exposes the smoker in general to higher levels of noxious agents than indicated by standard machine estimates.

Of the various reduction methods discussed, it appears that filter bidis and perforated bidis are more suitable for commercial adoption. In a randomised double blind trial, the cotton filter bidis were found to be acceptable to the smoker (23).

It needs to be emphasized that reduction methods for tar and nicotine are suggested not as an alternative to primary prevention programmes but as an adjunct to it.

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Agricultural Experiments to Reduce Toxic Substances in Cigarette Smoke

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The chemistry of tobacco leaf has a profound influence on the qualitative and quantitative composition of tobacco smoke and its biological effects. The leaf chemistry in turn is determined by the production practices and processing techniques. The chief among them are the genetic and cultural factors (agronomical, pathological, entomological and physiological) and finally the post-curing technologies. The work done on these three aspects, by the author and others in the field, is discussed in the present paper. Genetic modifications of tobacco plants have resulted in successfully evolving high yielding, pest and disease resistant varieties commensurate with desirable quality traits. However, very few deliberate attempts have so far been made to exploit the existing genetic variabilities in lowering tar levels. Other factors also have not yielded any tangible results. It needs to be added that spectacular results have been achieved by adopting innovative cigarette manufacturing technologies for producing low tar cigarettes.

Introduction

The first most authentic report was issued by the U.S. Surgeon General in 1964 (1) which evaluated the causal significance of the association of cigarette smoking and disease. Several other reports (2–4), have been published since then linking smoking to cancer as well as other diseases. The 1982 report (2) indicated cigarette smoking to be the single major cause of cancer mortality in the US, accounting for 30% of total mortality. The 1983 report (3) stated that cardiovascular diseases accounted for more deaths than any other smoking-related disease, including cancer. The 1984 report (4) concluded that cigarette smoking was the major cause of chronic obstructive lung diseases in both men and women.

It has been demonstrated that nicotine, carbon monoxide, hydrogen cyanide, volatile phenols, polycyclic aromatic hydrocarbons (PAH), acrolein and acetaldehyde contribute to the toxicity and/or carcinogenicity of tobacco smoke (5, 6). Correlation has been demonstrated between the above smoke constituents and leaf chemistry (7).

The chemical and biological characteristics of tobacco smoke are governed by the chemical and physical properties of tobacco in addition to the design and other physical parameters of the cigarette. The farm production practices are known to

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Abbreviations : cig : cigarette, TPM : Total particulate matter.

profoundly affect the physico-chemical characteristics of tobacco. The major parameters are 1) genetic factors 2) cultural practices and 3) post-curing processes.

In the present overview, the effects of the above factors are discussed in relation to smoke characteristics.

1. Genetic variability and smoke characteristics

So far tobacco breeding programmes have aimed at improving the agronomical characteristics, disease resistance and physico-chemical characteristics of tobacco. Very little deliberate attempt has been made to modify the tobacco genotypes to yield lower tar varieties. The main reasons for this lecuna are : 1) the standard smoke analysis requires large quantities of tobacco whereas the sample available for any entry in any breeding programme is usually small and 2) the genetic studies require large number of samples to be analysed for smoke characteristics.

Table 1. Smoke analysis of different cultivars grown in different agroclimatic zones of South India

	TPM mg/cig	Nicotine mg/cig	TPM per g dry tobacco smoked (mg)	Nicotine per g dry tobacco smoked (mg)
Hunsur				
1. 2359	29.6	2.0	52.8	3.5
2. FCV special	38.6	4.9	77.1	9.8
3. 1494	38.0	3.1	76.0	6.1
Rajahmundry				
4. CTRI special	29.6	2.3	52.2	4.1
5. CTRI Y_1 grade	34.2	2.2	61.3	3.9
6. CTRI Y_2 grade	35.0	2.4	69.5	4.7
7. CTRI LBY_1 grade	34.6	2.2	66.7	4.2
8. CTRI LBY_2 grade	31.8	2.1	61.7	4.1
9. Kanakaprabha	31.6	1.5	62.6	3.0
Devarapalli				
10. Jayasri	30.1	2.4	52.9	4.2
11. CTRI special	30.2	1.9	54.0	3.3
12. 16/103	32.1	2.3	51.5	3.6
Kandukur				
13. Jayasri	32.4	2.7	52.9	4.4
14. FCV special	31.1	2.2	53.4	3.8

There is enough evidence to show that tobacco genotypes differ in their potential to produce tar. Tso (8) reported on the range of variation in Wet Total Particulate Matter (WTPM) and nicotine in 6 flue-cured cultivars. While the range of WTPM was from 27.0 to 34.0 mg/cig.,the smoke nicotine varied from 1.3 to 3.0 mg/cig.

Legg and Chaplin (9) compared 5 flue-cured with 5 burley tobacco cultivars and did not find much difference in the range of total particulate matter (TPM) levels (Flue-cured : 32.8 to 39.8 mg /cig., Burley : 31.0 to 38.2 mg/cig.). However, tar levels (Flue-cured : 26.3 to 30.4 mg/cig., Burley : 22.0 to 28.8 mg/cig.), were lower whereas nicotine levels were higher (Flue-cured : 2.5 to 3.9 mg/cig., Burley : 3.5 to 4.2 mg/cig.), in burley tobacco compared to the flue-cured tobacco cultivars.

In smoke analysis of different tobacco cultivars grown in different agroclimatic zones of South India, we observed that TPM values ranged from 29.6 to 38.6 mg/cig., and nicotine levels varied from 1.5 to 4.9 mg/cig. (Table 1)

Differences in TPM and nicotine levels of different breeding lines of tobacco cultivars were also observed by us, depending on the kind of soil in which it was grown. Table 2 shows the smoke analysis of several varieties which were grown either in black or light soil. Generally, TPM and nicotine levels in cigarette smoke were observed to be lower when the tobacco was grown in black soil as compared to the same variety grown in light soil.

The smoke analysis data from a wide range of existing genetic material indicates that tobacco varieties differ, though not widely, in tar and nicotine

Table 2. Evaluation of smoke TPM and nicotine in some breeding lines grown in black and light soils

Variety	Soil	TPM mg/cig	TPM per g dry tobacco smoked (mg)	Nicotine mg/cig	Nicotine per g dry tobacco smoked (mg)
NC-12	Black	32.2	49.6	2.1	3.2
	Light	40.0	69.2	3.6	6.1
MDC 30	Black	35.1	60.3	2.0	3.5
	Light	38.9	76.3	3.9	7.5
46-4-3	Black	35.8	60.8	1.5	2.5
	Light	34.1	50.9	3.0	4.5
MDC 29	Black	32.0	61.3	1.7	3.2
	Light	34.8	55.3	3.9	6.2
F 220	Black	31.7	51.1	2.1	3.4
	Light	37.9	63.4	3.9	6.6

productions. This suggests the possibility of deliberate genetic modification to produce tobacco which can yield lower tar and nicotine, when smoked. Such an attempt was first made by Matzinger and Wernsman (10) in 1970. They created a broad-based genetic population by intermating eight black shank resistant flue-cured varieties. The range of WTPM per cigarette in these 160 full-sib families was found to be 25.2 mg to 36.6 mg. In an extension of this study (11) recurrent mass selection was initiated with 20 full-sib families with the lowest WTPM obtained from the previous study. To avoid the need for large quantities of tobacco and time and effort required for the standard method of smoke analysis, the particulate matter index (PMI) technique of Long and Wilson (12) was adopted in this study. Following three cycles of selection, the performance of the selected population showed a reduction of PMI from 210 to 180 mg/cig. Selection for lowest tar in cycle 3 resulted in concomitant reductions in leaf nicotine (3.5 to 2.4%) and smoke nicotine (3.6 to 2.5 mg/cig).

The current evidence suggests that tar levels in tobacco smoke can be reduced by genetic modification and the genetic correlation between tar and nicotine will result in simultaneous reduction in nicotine as well.

2. Cultural practices

a) Nitrogen fertilization : The various farm production practices may profoundly influence the physico–chemical characteristics of leaf tobacco which ultimately affect the smoke chemistry and its biological properties. Tso (8) reported the effect of nitrogen fertilization, sucker control methods, ripeness and rate of curing on the smoke characteristics. It was observed that burley tobacco at higher rate of nitrogen fertilization showed more weight of tobacco per cigarette and correspondingly more TPM and nicotine per cigarette, whereas in flue-cured tobacco the higher rate of nitrogen fertilization resulted in lesser weight of cigarette and lower levels of

Table 3. Effect of cultural practices on tobacco smoke composition *

	TPM mg/cig	TPM per g dry tobacco smoked (mg)	Nicotine mg/cig	Nicotine per g dry tobacco smoked (mg)
Nitrogen levels				
30 kg N/ha	28.8	49.4	2.1	3.6
45 kg N/ha	29.8	50.1	2.5	4.2
60 kg N/ha	32.8	59.4	3.2	5.8

N/ha : Nitrogen per hectare;
* Source : Kameswara Rao et al (14)

TPM but higher levels of nicotine. Yoshida (13) reported similar results for flue-cured tobacco i.e. as the rate of nitrogen fertilization increased from 5 to 20 kg per 10 acres, a decrease was observed in the tar levels of smoke of cigarettes-particularly in those made of tobacco leaves of middle position-however the nicotine levels increased with increasing use of nitrogen fertilizer. We (14) have observed increasing TPM as well as nicotine levels in cigarette smoke with increasing use of nitrogen fertilizer. (Table 3)

b) Effect of degree of ripeness: The effect of degree of ripeness on smoke characteristics is shown in Table 4.The TPM levels increased substantially with increasing leaf maturity in Yoshida's (13) experiment as well as Tso's (8) experiment. We (14) have also observed higher TPM levels in smoke of cigarettes made of mature leaves compared to immature leaves though no further increase was observed with over maturing. Degree of ripeness did not show any substantial effect on the nicotine delivery per cigarette in these experiments.

Table 4. Effect of degree of ripeness on the smoke characteristics[+]

	Tso		Yoshida*		Kameswara Rao et al			
	TPM mg/cig	Nicotine mg/cig	Tar mg/cig	Wt./cig (g)	TPM mg/cig	TPM per g dry tobacco smoked (mg)	Nicotine mg/cig	Nicotine per g dry tobacco smoked (mg)
Immature	24.0	2.3	24.7	1.04	21.7	44.9	1.9	3.9
Mature	25.0	2.5	26.1	0.95	24.2	48.4	2.1	4.2
Over mature	26.0	2.6	29.0	1.10	22.1	46.2	2.0	4.2

* Averaged over values of two leaf positions

[+] Source : Tso (8), Yoshida (13) and Kameswara Rao et al (14)

c) Effect of topping: In tobacco production, decapitation of the plant is an essential practice to obtain leaves of the required physico-chemical characteristics. The results reported by Yoshida (13) showed that cigarettes made of tobacco from untopped plants delivered less tar to the smoke than topped plants (Table 5). Similar results were obtained by us (14). The difference, however was not substantial probably because of late topping.

d) Effect of sucker control materials: As a consequence of topping operation auxilliary buds, called suckers, grow and reduce the yield and quality of tobacco. To arrest the sucker growth a number of chemicals are found to be effective. Some of them which are systemic may bring about further metabolic changes which may affect the leaf and smoke characteristics.

Tso (8) reported on the effects of different sucker control agents as compared to hand suckering method on the smoke characteristics. In burley tobacco the hand suckered control delivered the lowest TPM (22 mg/cig) and nicotine (2.9

Table 5. Effect of topping on the smoke characteristics [+]

	Yoshida		Kameswara Rao et al	
	Tar [*] mg/cig	Nicotine mg/cig	TPM mg/cig	Nicotine mg/cig
Topped	26.2	—	23.0	2.0
Untopped	21.0	—	22.3	2.0

[*] Averaged over 6 values for different leaf position
[+] Source : Yoshida (18) and Kameswara Rao et al (14)

mg/cig) levels and closely hand suckered tobacco gave the highest tar yield (25 mg/cig) and nicotine (3.8 mg/cig) levels, while the four chemical sucker control agents (methyl caprate, methyl pelargonate, octanol and decanol) had intermediate effects. In flue-cured tobacco the use of octanol-decanol mixture delivered the lowest TPM (26.5 mg/cig) and nicotine (2.6 mg/cig) level and again the closely hand suckered tobacco gave the highest TPM (33.0 mg/cig) and nicotine (3.9 mg/cig) delivery.

e) Effect of stalk position : The chemical characteristics of tobacco leaf differs according to the position on the stalk substantially. Darkis et al (15) and Tso (16) studied comprehensively the effect of varieties and stalk positions on a number of smoke constituents including TPM, nicotine, pH, carbon monoxide, carbon dioxide, acetaldehyde, acrolein, hydrogen cyanide, phenols, benzo (a) pyrene and benz (a) anthracene. It was noticed (Table 6) that irrespective of the variety, TPM and nicotine levels in smoke increased with ascending stalk positions.

Table 6. Effect of stalk position on the smoke characteristics [+]

Stalk Position	SC 58		LN 38		C 139		NC 95	
	TPM mg/cig	Nicotine mg/cig	TPM mg/cig	Nicotine mg/cig	TPM mg/cig	Nicotine mg/cig	TPM mg/cig	Nicotine mg/cig
1.	20.8	0.9	19.7	0.2	17.3	0.4	17.2	0.8
2.	27.8	1.1	15.1	0.1	16.6	0.4	23.3	1.0
3.	30.8	2.3	27.6	0.3	15.4	0.6	33.0	2.2
4.	39.8	4.4	28.0	0.3	27.5	1.1	34.0	3.2
5.	40.5	4.8	31.4	0.3	32.6	1.9	38.9	3.7
6.	44.4	5.6	37.3	0.9	36.4	2.8	41.7	4.6
7.	46.4	6.7	32.5	0.3	40.2	3.1	45.6	4.4
8.	53.8	8.0	35.2	0.4	46.2	3.2	48.9	4.9

[+] Source : Tso et al (16)

3. Post-curing processes

a) Effect of ageing: After the completion of curing and bulking, flue-cured tobacco is subjected to 'ageing' process which is equivalent to 1 to 2 years of storage under ambient conditions or controlled conditions of temperature and humidity. Ageing is meant to make the tobacco mellow and fit for smoking. Many physico-chemical changes like reduction in nicotine content occur during ageing (17, 18).

We studied the changes in smoke characteristics during ageing of flue-cured tobacco for 24 months. The data in Table 7 show about 26.5% reduction in TPM per cigarette and 54.3% reduction in nicotine per cigarette after 24 months of ageing.

Table 7. Variation in tobacco smoke composition with ageing[+]

Sample	TPM mg/cig	TPM per g dry tobacco smoked (mg)	Nicotine mg/cig	Nicotine per g dry tobacco smoked (mg)
1. Immediately after redrying	32.1	58.4	3.5	6.4
2. 3 months after redrying	29.6	52.6	2.2	3.9
3. 6 months after redrying	29.3	53.4	2.1	3.8
4. 12 months after redrying	28.4	47.9	2.1	3.4
5. 18 months after redrying	29.2	51.7	2.0	3.6
6. 24 months after redrying	23.6	43.2	1.6	3.0

[+] Source : Kameswara Rao et al (19)

b) Effect of additives: Salts like mono ammonium phosphate, sodium and potassium citrate, sodium acetate, potassium nitrate and potassium and sodium tartarates are added to cigarette paper to modify the colour of cigarette ash and to enhance combustion which in turn reduces tar yields substantially (20). Treating the tobacco with some salts and oxidizing agents like hydrogen peroxide or ozone (21, 22) is also known to reduce tar and nicotine deliveries in a cigarette.

We studied the effect of treating tobacco with potassium citrate, potassium

ascorbate and hydrogen peroxide on the levels of TPM and nicotine in cigarette smoke (Table 8). Maximum reduction in TPM per cigarette was recorded with potassium citrate as the additive (35%) followed by hydrogen peroxide (33%) and potassium ascorbate (28%) – all at highest concentrations. These additives also reduced nicotine levels by 23%, 38% and 27% respectively.

Table 8. Effect of additives on smoke constituents

Treatment	TPM mg/cig	TPM per g dry tobacco smoked (mg)	Nicotine mg/cig	Nicotine per g dry tobacco smoked (mg)	Hist$^+$ Rev. per plate
1. Citric acid 1%	33.7	70.4	2.8	5.9	—
2. Citric acid 2%	28.5	66.2	2.2	5.1	—
3. Citric acid 3%	28.3	61.7	2.1	4.7	—
4. Ascorbic acid 1%	36.7	76.7	3.1	6.6	—
5. Ascorbic acid 2%	35.8	81.4	3.0	6.8	—
6. Asocorbic acid 3%	33.9	70.1	3.1	6.3	—
7. Potassium ascorbate (5.5%)	31.9	54.7	2.4	4.1	—
8. Potassium ascorbate (11.0%)	25.9	48.1	2.2	4.1	—
9. Potassium ascorbate (16.5%)	23.3	42.2	1.9	3.4	—
10. Potassium citrate (2.77%)	28.8	51.6	2.5	4.4	84 ± 11
11. Potassium citrate (4.54%)	25.5	50.3	2.2	4.3	80 ± 12
12. Potassium citrate (8.31%)	21.0	36.9	2.0	3.4	31 ± 11
13. Hydrogen peroxide 10%	27.2	49.5	2.2	3.9	73 ± 11
14. Hydrogen peroxide 30%	21.7	41.1	1.6	2.9	20 ± 2
15. Control	32.4	65.4	2.6	5.3	157 ± 19
16. B(a)P	—	—	—	—	360 ± 40

It is seen from the table that higher the concentration of the additives, the greater the reduction in smoke parameters viz., TPM and nicotine. Furthermore, these reductions seem more to be due to potassium ion than citrate or ascorbate ions. The mutagenicity data were available for potassium citrate and hydrogen peroxide only. Reduction in the revertants per plate was over 80% at the highest

concentrations of these additives, thus leading to substantial reduction in tumorigenic potential.

Besides the above mentioned agricultural practices for reducing harmful components of tobacco smoke, changes in cigarette manufacturing technology over the last 30 years has also helped in dramatically reducing tar and nicotine levels in the smoke of the cigarettes manufactured in the West.

In conclusion, it may be stated that it should be possible for us in India also to produce low tar–low nicotine cigarettes by selecting the right type of tobacco and adopting innovative cigarette manufacturing technologies.

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Studies on Tobacco Specific Nitrosamines and Other Carcinogenic Agents in Smokeless Tobacco Products

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Analytical and biological studies of smokeless tobacco products were carried out. Tobacco with or without betel quid, masheri, a pyrolysed tobacco product used as dentifrice, creamy snuff and snuff used for inhalation were analysed for tobacco specific nitrosamines (TSNAs). All the products contained µg/g quantities of nitrosonornicotine (NNN), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), N'-nitrosoanatabine (NAT) and N' -nitrosoanabasine (NAB) when analysed. Even the tobacco used for preparation of cigarette, bidi and chutta had substantial amounts of TSNAs. In short term tests for mutagenicity of tobacco, masheri and two important TSNAs e.g. NNN and NNK, it was observed that all were mutagenic in Ames test and micronuclei test. Subsequent long term studies on carcinogenicity revealed that masheri induced preliminary stomach papillomas, while tobacco, NNN and NNK induced lung tumors, forestomach tumors and occasionally liver tumors. The studies conclusively prove that tobacco products containing preformed TSNA are mutagenic and carcinogenic.

Introduction

In developed countries, cigarette smoking accounts for most of the tobacco use, while in India a wide spectrum of tobacco habits is observed. Chewing tobacco with lime or betel quid, use of snuff for inhalation or as a dentifrice or as a substitute for tobacco chewing are some of the tobacco habits which employ smokeless tobacco. Smoking cigarettes, bidis,chuttas, chillum, hookah, hookly are part of the smoking habits practised in different parts of the country (1). Association of tobacco habits with oral cancer was reported by Niblock in 1902 (2) and since then several epidemiological studies have supported this contention (3,4). Voluminous literature pertaining to toxic chemicals in cigarette tobacco and

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Abbreviations : NNN : N'-nitrosonornicotine, NNK : 4-(N-methyl-N-nitrosamino) -1-(3-pyridyl)-1-butanone, NAT : N-nitrosoanatabine, NAB : N'-nitrosoanabasine, NPYR : N-nitrosopyrrolidine, NDMA : N-nitrosodimethylamine, TSNA : Tobacco-specific N-nitrosamines, PAH : Polycyclic aromatic hydrocarbons.

cigarette smoke is available (5–7) but relatively very little data on toxicants in smokeless tobacco are at hand. Early efforts using several solvent extraction procedures to indentify carcinogenic agents in chewing tobacco (Wadakhan variety), were not so successful as skin application of these extracts yeilded mainly hyperplasia (8) and few squamous cell carcinomas at the site of application (9).

Since last few years we have taken up the studies on smokeless tobacco and have tried to identify the toxic/carcinogenic principles present in chewing tobacco, snuff, masheri etc. A special emphasis has been laid on the analysis of volatile and tobacco specific nitrosamines (TSNA) which are reported to be present in cigarette smoke (10). Simultaneously we have tested the mutagenicity and carcinogenicity of chewing tobacco and masheri.

Following these studies we have analyzed the saliva of tobacco habituees as it is a probable link between tobacco and the target organ namely oral cavity.

Materials and methods

Tobacco products

1. Dry processed tobacco leaves of two species. *N.rustica* and *N.tabacum* were obtained from Tobacco Research Station, Gujarat Agricultural University, Anand.
2. Popular brands of inhalation snuff and creamy snuff tooth paste were purchased from local market at Bombay.
3. Masheri : *N.tabacum* variety of tobacco was purchased from the local market and roasted on an iron plate at 150°C and 180°C respectively to obtain the brown and black variety of masheri. Both the varieties are used as dentifrice as per the taste of the user. Many women use it frequently as a substitute for tobacco chewing.
4. Betel quid + tobacco : Freshly prepared betel quid consisting of two betel leaves, lime, catechu, betel nut and approximately 500 mg tobacco *(N.rustica)* was purchased from local vendors of betel quid and tobacco.
5. Tobacco from bidis, cigarettes and chutta : Popular brand of bidi, an indigenous substitute for cigarette, cigarette made in India and chutta an indigenous form of cigar, largely used by people from South-East India, were purchased in the local market.

Extraction and analysis of TSNAs namely nitrosonornicotine (NNN), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), nitrosoanatabine (NAT) and nitrosoanabasine (NAB) was carried out according to the procedure reported earlier (11).

Extraction and analysis of PAH profile was done as described earlier (12).

Mutagenicity studies

Tobacco extract : To 50 g of commercial dry tobacco *(N.tabacum)* was added 100 ml ethanol and kept at room temperature with occasional stirring. The alcoholic extract was then filtered and lyophilised. The dry residue was then

dissolved in 10 ml distilled water and this stock solution was used for testing mutagenicity.

Masheri extract : Thirty grams of brown or black masheri was extracted with 500 ml of toluene in a Soxlet apparatus for 6-8 hours. The extract was then subjected to flash evaporation to get a dry residue, which was dissolved in DMSO and diluted with distilled water to obtain desired concentration.

Ames test : This was carried out in histidine deficient *S.typhimurium* strains TA 98 in presence of metabolic activation as described by Ames et al (13).

Micronuclei test : Induction of micronuclei in bone marrow cells of Swiss male mice was studied using Schmid's procedure (14).

Carcinogenicity studies

Animals : Eight weeks old male Swiss strain mice were obtained from the animal colony of Cancer Research Institute, Bombay. They were housed in steel cages (4 per cage) and kept at 20-21°C and 50-55% humidity. Food and water were given ad libitum.

Tobacco extract in diet : Fifty grams of commercial tobacco *(N.tabacum)* was extracted with 300 ml of ethanol at room temperature for 24 hours using a mechanical shaker. The alcoholic extract was then mixed with 5 kg of standard diet (15). The mixed diet was spread for few hours to allow excess alcohol to evaporate and stored in cold room. Fresh diet was prepared every two weeks. Eight weeks old Swiss male mice were put on this diet throughout the life and were allowed to eat ad libitum. Animals were killed when moribund.

Treatment of 10% masheri diet : Black and brown masheri were directly mixed with standard diet, so as to obtain a 10% concentration in the diet. Eight weeks old Swiss male mice were put on this diet throughout life and were allowed to eat ad libitum. Mice were killed when moribund. Mice kept on standard diet served as controls for both the groups.

Treatment of NNN by gavage : Eight weeks old Swiss male mice were treated with NNN (1 mg/day/mouse) five days a week by gavage, till they received a total dose of 22 mg. After discontinuation of the treatment mice were kept under observation till death.

Treatment of NNK by gavage : Eight weeks old Swiss male mice were administered NNK (1 mg/day/mouse) five days a week by gavage. They received a total dose of 22 mg, after which treatment was discontinued and animals were kept under observation till death. Mice receiving distilled water served as controls.

Mice, dead or sacrificed were dissected carefully for any gross abnormality or tumor and fixed in 10% neutral formalin. Tissues were processed by routine procedures for histopathology and observed under the microscope.

Field studies : Groups of 7 to 10 healthy subjects with tobacco chewing or masheri habits belonging to both sexes and between the ages of 25 to 50 years

were selected for analysis of nitrosamines in saliva. Saliva (20 ml) was collected as per the procedure described earlier (16). As controls, saliva of non-chewer males (n = 10) and females (n = 4) of comparable ages were analyzed.

Observations

Two species of tobacco, *N.tabacum* and *N.rustica* as well as tobacco with betel quid were analyzed for TSNA content. Table 1 shows the values of NNN, NNK, NAT, or NAT + NAB. The resolution of NAB peak was not achieved satisfactorily hence values for NAB individually are not shown but are given in combination with NAT. It is evident that *N.rustica* variety of tobacco is considerably richer with respect to all nitrosamines compared to *N.tabacum*. It may be noted that *N.rustica* is favoured more by tobacco chewers. Tobacco + betel quid also contains significant amount of TSNAs.

Table 1. TSNA in tobacco and tobacco with betel quid

Tobacco species	Betel quid	TSNA (μg/g dry wt.)			
		NNN	NNK	NAT	NAT + NAB
N.Tabacum	-	1.50	0.52	ND	1.03
N.Rustica	-	14.96	25.77	ND	78.17
N.Rustica	+	26.80	4.90	23.20	ND

ND : Not done

Creamy snuff used as tooth paste and snuff used for sniffing/inhalation and black/brown variety of masheri were analyzed for TSNAs. Use of masheri begins at a very early age as a dentifrice and is used by women (low income group) frequently as a substitute for tobacco chewing. All the four tobacco products contain TSNAs, in substantial amount, as seen in Table 2.

We have then analyzed tobacco used in the preparation of bidis, cigarettes and chuttas. Bidis and cigarettes are smoked all over India while chuttas are smoked by rural women and men from south eastern part of India. From Table 3 it may be noted that though tobacco of the same species *(N.tabacum)* is used for manufacture of all the three products, TSNA levels in chutta tobacco are highest. It is therefore important to see if soil and climate affect TSNAs since chutta tobacco is grown in rich black soil of eastern Godavari delta.

Since masheri and snuff are pyrolysed tobacco products, we analyzed snuff and black masheri for carcinogenic PAH types of compounds (12). Table 4 shows that both the tobacco products are rich in carcinogenic PAH.

Following analytical studies, we tested extracts of chewing tobacco, masheri, NNN and NNK for mutagenicity using Ames test. All the four test substances show a dose dependent increase in the his^+ revertants in presence of metabolic activation as seen in table 5.

Table 2. N-nitrosamines in snuff (sniffing), creamy snuff (tooth paste) and masheri

Tobacco product	N-Nitrosamines (μg/g)				
	NNN	NAT	NNK	NPYR	NDMA
Snuff (sniffing)	1358.83	1856.50	245.46	ND	ND
	136.98	113.06	110.68	ND	ND
Brown masheri	1.05	0.98	0.41	0.03	0.01
Black masheri	1.05	0.78	0.42	0.03	0.05
Creamy snuff	48.07	26.62	8.83	ND	ND
	12.78	10.72	10.99	ND	ND
	19.48	11.09	9.97	ND	ND
	12.95	10.80	12.54	ND	ND

ND : Not done

Table 3. Tobacco specific nitrosamines in Indian tobacco used in cigarette, bidi and chutta

Type	Sample	Nitrosamines (μg/g)		
		NNN	NAT	NNK
Cigarette	1	58.0	15.1	4.8
	2	1.3	0.8	0.4
Bidi	1	6.2	9.0	0.4
	2	12.0	12.5	1.4
Chutta	1	295.8	686.7	210.3

Table 4. Carcinogenic and co-carcinogenic polycyclic aromatic hydrocarbons in masheri and snuff used for inhalation

Compounds	In masheri	In snuff	
		Sample 1	Sample 2
	(μg/g)	(μg/g)	(μg/g)
Benzo (a) pyrene	0.012	0.066	0.008
Benzo fluoranthenes	0.035	0.231	0.043
Benzo (a) anthracene	0.079	0.049	0.019
Chrysene and triphenylene	0.192	0.121	0.037
Benzo (e) pyrene	0.017	0.110	0.010
Pyrene	0.169	0.091	0.060
Fluoranthene	0.218	0.105	0.055
Benzo perylene	0.009	0.017	0.006
Perylene	0.004	0.050	0.002
Benzo nepthol thiophene	0.057	0.089	0.011

Table 5. His^+ revertants in *S.typhimurium* strains TA 98 and TA 100 induced by tobacco masheri and TSNAs in presence of metabolic activation

	His^+ revertants / plate				
Concentration in μg/plate	Tobacco extract (TA 98)	Masheri extract (TA 98)		TSNA (TA 100)	
		Brown	Black	NNN	NNK
0	50	45	ND	123	ND
25	168	235	341	ND	ND
50	198	298	400	108	125
100	234	448	504	171	195
250	ND	ND	ND	272	320
500	ND	ND	ND	370	499

ND : Not done

Results are mean of eight petri plates. Lyophilised tobacco and masheri extracts were dissolved in known quantity of DMSO. B(a) P (5μg/plate) was used as a positive control; His^+ revertants per plate were 900.

To establish mutagenic potential of a test substance, it is important to test it in at least two short term tests, one bacterial and the other mammalian. We

therefore tested these four substances in micronuclei test in Swiss male mice. (Table 6)

Both, tobacco products and their important TSNAs namely NNN and NNK induce significant increase in micronucleated cells when compared with the control group as seen in Table 6.

Table 6. Induction of micronuclei in the bone marrow cells of swiss mice by tobacco, masheri and TSNAs

Test substance	Concentration	% of micronucleated polychromatic erythrocytes	P/N Ratio
Distilled water		0.27 ± 0.03	0.998
Tobacco	0.25 mg per mouse	1.09 ± 0.05*	1.050
Brown masheri	0.05 mg per mouse	0.93 ± 0.03*	0.960
Black masheri	0.05 mg per mouse	1.05 ± 0.03*	0.982
NNN	0.25 mg per g body wt.	1.00 ± 0.05*	0.998
NNK	0.25 mg per g body wt.	1.30 ± 0.05*	0.997

* $P < 0.05$ (Compared with distilled water group)

P/N : Ratio of polychromatic to normochromatic cells (Ratios nearing unity indicate nontoxicity of the dose)

Along with short term studies for mutagenicity, long term studies on carcinogenicity of chewing tobacco, masheri, NNN and NNK were initiated in Swiss male mice, as per details given in Materials and Methods section. It is evident from Table 7 that both NNN and NNK are potent carcinogens and induce 80-100% tumours in lung, liver and/or stomach. Tobacco extract induced only lung tumours (41%). Masheri is conceivably a weak carcinogen since it largely induces stomach papillomas (53%-56%) in treated mice.

Following the experimental studies we initiated a small field study to assess the possible exposure to TSNAs in people practising tobacco habits (16).

It is interesting to observe (Table 8) that all the subjects with different tobacco habits had detectable levels of TSNAs in ppb quantities while only 1 male and 1

Table 7. Tumour incidence in swiss male mice treated with TSNAs by gavage, tobacco extract and masheri in diet

Test	Age in Months	Tumour Type					
		Lung	Liver	Stomach papilloma	Liver + stomach papilloma	Lung + stomach papilloma	Total incidence
Control	18–25	2		1			3/27 (11%)
Tobacco	21–25	7					7/17 (41%)
10% Brown masheri	19–25			7	3		10/18 (56%)
10% Black masheri	19–25			10			10/19 (53%)
NNN	17–25	8	1	3	1	2	15/18 (83%)
NNK	17–25	14	3				17/17 (100%)

Table 8. Total N-nitrosamines in the saliva of habitual users of tobacco with lime/betel quid and masheri.

Tobacco use	Sex	No. of subjects	Total TSNAs in saliva (ppb) (range)	
Tobacco + lime	M	10	165 ± 61	(22–600)
Tobacco + betel quid	M	7	63 ± 35	(26–129)
Tobacco + betel quid	F	10	63 ± 48	(6–128)
Masheri	F	9	61 ± 30	(14–121)
None	M	10	—	(0– 20) *
None	F	4	—	(0– 30) *

* Nitrosamines were detected in one sample of each group.

female non-chewer (out of a sample of 10 and 4 respectively) had detectable levels in their saliva. Probably these two individuals did not admit having tobacco habit. It is also noteworthy that there is a wide range in TSNA content of saliva of the

subjects using same type and same amount of tobacco/masheri. These observations are in agreement with previous reports (17,18).

Discussion

Previous reports from our laboratory as well as other laboratories have clearly shown that smokeless tobacco (chewing variety) and snuff (dipping variety) contains TSNAs in μg/g quantities (19–21). In this paper we are reporting presence of TSNAs in different commercial tobacco products such as creamy snuff, masheri and inhalation snuff, many of which are used by lay people as innocuous products from a very early age. We have further shown that the TSNAs, NNN and NNK are potent mutagens and carcinogens. Except for few studies (22–24), mutagenicity of TSNAs is not reported widely but the carcinogenic potential of NNN and NNK has been established in rats and hamsters (25–27) and our present data are in agreement with these reports. We have also shown here that chewing tobacco and masheri are mutagenic. Since both these habits are practised by a large segment of society the possible mutagenic load on the population needs to be carefully assessed.

As for the carcinogenic effect of chewing tobacco and masheri, we have observed a significant number of tumours in treated animals. In earlier studies mice treated with solvent extract of tobacco developed few tumours (28), but these studies did not establish unequivocally the oncogenic potential of chewing tobacco. However, recent reports on carcinogenicity of chewing tobacco and snuff used for dipping have firmly established the carcinogenic effect of both these products (27,29). Present work further supports the fact that tobacco used for chewing, as well as masheri possesses carcinogenic potential. It is also interesting to note that the saliva of people chewing tobacco with lime or betel quid or using masheri contains a considerable amount of TSNAs which are in direct contact with the oral mucosa, a target tissue of tobacco related cancer. The TSNAs present in ppb quantities in the course of a single chew will amount to mg quantities in course of time, in an individual who chews tobacco 10 times or more per day. Since nitrosamines are proven carcinogens in about 40 species (30), it is quite likely that these TSNAs induce malignancy at the site of contact. Further, it is interesting to observe that few individuals had relatively much higher levels of TSNAs as compared to others in the same group. It is probable that the quantity of TSNAs present in saliva of an individual may be one of the determinants of developing oral cancer.

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Relevance of Low Tar-Low Nicotine Cigarettes and Bidis in the Indian Situation

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Introduction

Epidemiologic studies in India have demonstrated cigarette and bidi smoking to be risk factors in cancers at several sites, coronary heart disease and chronic obstructive lung disease (1, 2, 3). As yet there are no organised smoking control programmes or any legislation to limit the tar and nicotine yield. However, in recent years there is a greater awareness of the hazards of smoking. Government has restricted tobacco advertising and is poised to formulate a smoking control programme which would aim at dissuading nonsmokers (especially adolescents) from taking up smoking besides encouraging smokers to quit smoking (4). The question arises whether setting limits to tar and nicotine yields of cigarettes and bidis should also be a component of a comprehensive smoking control strategy in the country.

Tar and nicotine levels

Cigarettes in the west

Following the Reports of Royal College of Physicians in U.K. (1962) (5) and U.S. Surgeon General's Advisory Committee (1964) (6) which showed beyond doubt that smoking was causally associated with cancer at a certain anatomical sites, coronary heart disease and chronic obstructive lung disease, the U.S. Public Health Servies (7) reviewed the issue of smoke constituents and stated that "the preponderance of scientific evidence strongly suggests that lower the 'tar' and nicotine content of cigarette, the less harmful would be the effect". In the following decades, Government in several western countries pursued the policy of limiting tar and nicotine levels and the cigarette companies had to modify their product accordingly. In the US in 1954, the average "tar" yield of the sales weighted average cigarette was 37 mg and average nicotine 2.6 mg and in 1980, the comparable figures were less than 14 mg "tar" and 1 mg nicotine (8).

Indian cigarettes

Levels of tar and nicotine in some popular brands of cigarettes marketed in

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Table 1. Levels of tar and nicotine in cigarettes[+]

Type	Number of brands tested	Range of values	
		Tar mg/cigarette	Nicotine mg/cigarette
Plain (70 mm)	3	21.16 to 21.94	1.04 to 1.21
Filter tip (70 mm)	3	18.33 to 22.61	0.94 to 1.18
Filter king (35 mm)	5	24.19 to 28.27	1.23 to 1.78

[+] Estimated under standard international conditions, in popular brands marketed in 1979-80.

the early eighties are shown in Table 1 (9). These yields are akin to those of the cigarettes marketed in the west in the sixties (8). Furthermore, it is of importance to note that filter tip cigarettes do not necessarily have lower levels of tar and nicotine than plain cigarettes and filter king cigarettes have much higher levels compared to both plain and filter tip cigarettes. Thus filter cigarettes in the country do not connote low tar and low nicotine cigarettes but mislead the smoker into thinking so. The filter efficiency was found to be less than 20% in the Indian cigarettes whereas it is greater than 30% in the west (9). This is mainly because the filters used are short (10–12 mm long) compared to those in the west (greater than 20 mm).

It may also be worth noting that levels of other noxious substances like hydrogen cyanide, (366–638 μg /cigarette) phenols (118–226 μg/cigarette) and benzo(a)pyrene (85–114ng/cigarette) in the currently marketed Indian cigarettes are higher (10) than those of plain cigarettes marketed in US over a decade ago (11).

Bidis

Levels of tar and nicotine in the bidis which are either 60 mm or 80 mm long are high (9). Direct comparisons of the machine estimates with those of cigarettes are not appropriate as the bidis have to be smoked at a puff frequency of 2 puffs per minute instead of the international standard of one, to keep them burning. However, machine estimates based on smoking behaviour of bidi smokers (5 puffs/min., 30 mm butt length) and cigarette smokers (2 puff/min, 23 mm butt length) which is indicative of exposure levels in the smokers could be meaningfully compared. These estimates (Table 2) show that smokers of long bidis are exposed to higher levels of tar and nicotine than smokers of all types of cigarettes whereas smokers of regular bidis are exposed to higher nicotine than smokers of plain and filtertip cigarettes but lesser tar compared to smokers of any cigarette (10). The only brand of filter bidis which is manufactured for export purposes has even

Table 2. Emission levels in a selected brand of bidis and cigarettes based on smoking behavior of smokers[+]

Type of smoke	Tar mg/stick	Nicotine mg/stick
Bidi		
Regular	25.5	2.5
Long	46.5	3.7
Cigarettes		
Plain	36.5	1.9
Filter tip	32.0	2.0
Filter king	41.7	2.7

[+] Bidi smokers : puff frequency = 5 puffs/ min; butt length = 30 mm
Cigarette smoker : puff frequency = 2 puffs/ min; butt length = 23 mm.

higher levels of tar and nicotine compared to long bidis under standard smoking conditions (as modified for bidi) (9).

Health-effects of low yield cigarettes

As there are no low tar cigarettes or bidis, health effects of low yield products cannot be assessed in India. However, in the west several studies have addressed themselves to this question and their findings in brief are given below.

Cancer

Smokers who started smoking with filtertipped cigarettes and those who switched to low tar from high tar have been shown to have a lower risk of cancers of lung and larynx. In case control study, life long smokers of filter cigarettes had a 30 to 40% (12) and 25 to 49% (13) reduction in risk of lung cancer and laryngeal cancer respectively.

Coronary heart disease (CHD)

The risk of CHD in smokers is dose-dependent, higher the frequency of cigarettes smoked, higher the risk, and cessation of smoking decreases the risk (14). These findings indicate that the risk associated with lower yield cigarettes would be lower. However, epidemiologic studies have not shown consistent results. One study has shown a decrease in risk (15) whereas others have not (16). There is even one study which has shown an increase (17).

The constituents suspected to be of etiologic importance in coronary heart disease are nicotine and carbon monoxide. Low tar cigarettes generally have low nicotine but carbon monoxide may be unrelated to the levels of tar (18). It is possible that lack of unequivocal results observed in CHD are due to the fact that not all the relevant constituents of smoke, which are implicated in the pathogenesis of coronary heart disease are taken into account simultaneously in evaluating the risk.

Chronic obstructive lung disease (COLD)

Although the causal association of COLD to cigarette smoking is well documented, the components in the smoke responsible for higher risk are not known. Furthermore, there are very few studies on risk related to tar levels. In low yield cigarette smokers, one study has reported a nonsignificant reduction of deaths due to emphysema (19) and another a decrease in phlegm produced (20).

Overall mortality

It has been reported that those smoking lower tar and nicotine cigarettes showed a 15% decline in mortality rate (all causes) compared to those smoking high levels (14). This reduction in overall mortality, which could be considered as a composite index for assessment of health risk, clearly indicates that low tar and low nicotine cigarettes are less hazardous.

Doubts/reservations regarding promotion of low yield cigarettes

The US Public Health Service in 1979 (14) evaluated the available evidence regarding the health effects of low yield cigarettes. The report, while confirming the earlier conclusion, warned that the smokers would not derive benefit from low tar-nicotine cigarettes if they changed to higher number of cigarettes or to deeper inhalation. Furthermore, it emphasized that the most effective way of reducing the health hazards was to quit smoking. The 1981 report again confirmed the earlier evaluation and urged further work on identifying other (18) noxious agents of etiologic importance in the various smoking related diseases. (8)

The issues which have prevented an unqualified support for recommending low yield cigarettes are the following :

a) Compensatory smoking : Smokers who switch on to low yield cigarettes are suspected to change their smoking behaviour resulting in increase in frequency of number smoked and deeper inhalation.

b) Encouragement to smokers : The policy recommending low yield cigarettes may be misconstrued to mean low tar cigarettes are safe and free of hazards and might lead to encouragement to the smoker.

c) Self defeating policy : The policy might be self defeating as it provides an opportunity to cigarette companies to continue their vigorous promotional campaigns.

d) Changing cigarette : There is no evidence to suggest that the benefits of reduction of high yield to low yield would also be seen in the reduction from low yield to ultra low yield. Furthermore, there is no assurance that cigarette engineering would not introduce other compounds equally harmful or more harmful in the new cigarettes.

Relevance to the Indian situation

In India, where 76% of the population is rural, there is as yet hardly any awareness of hazards of smoking. Smoking cessation programmes, which need a more personalized approach than educational programmes would have to reach over a 100 million smokers in the subcontinent. Reaching the goal of a nonsmoking culture and a nonsmoking generation would be a gradual process to be achieved with continued education. In the interim, efforts to lower the risk of smoking related diseases could well include introduction of low tar-low nicotine cigarettes. No doubt some may not benefit from the policy, due to compensatory smoking behaviour. No doubt, some may feel encouraged to smoke. No doubt cigarette companies may try to wrest advantage in their promotional efforts. It seems these consequences of pursuing a low yield policy are exaggerated as compensation is suspected to be only partial (those who switched to low yield cigarettes were more likely to be ex-smokers(18).The outcome of the policy of low yield cigarettes followed assiduously in US and UK (both by governments and cigarette companies) has lead to an overall reduction in smoking rates. The advantage,cigarette companies (or bidi manufacturers) may gain,probably can be stemmed by adequate legislation. Finally, it is of importance to recognize that the issue in India is far more basic and is vastly different from the west where the issue is of shifting from low tar to ultra low. As of now, low yield cigarettes and bidis are nonexistent in India. Thus, none of the doubts raised against a low yield policy need deter India from pursuing it. In fact, research laboratories must be encouraged to device simple and inexpensive reduction methods for tar and nicotine in the bidis, characterise the bidi smoke and pass on the simple reduction technology to the bidi industry which has neither the know-how nor the infrastructure to test modified bidis. Besides being of help to the cottage industry, this approach has the advantage of health planners knowing exactly what is smoked by the majority of smokers in the country (bidi smokers are five times as many as cigarette smokers). It may be mentioned here that two laboratories have already put forth some reduction methods, (21–23). However, manufacturers have not been motivated to adopt them.

As far as the cigarette companies are concerned they have the technology to reduce the tar and nicotine to any desired level although they have not made any efforts to introduce them in India. It is also known that poorer quality tobacco is dumped in the third world countries as they have not been alert.

In the final analysis, there certainly is sufficient justification to initially limit the tar and nicotine levels to 15 mg and 1 mg respectively, as has been done in several countries. While recommending this policy, it needs to be emphasized, as

outlined in the WHO report (25) that "programmes to reduce tar in cigarettes and other product modification activities should not be seen by governments or tobacco companies as a justification for inaction on other antismoking activities – such as passing legislation against promotion of tobacco products, educating the public and starting smoking control programmes".

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Section III

Intervention Studies and Behaviour Modification

Tobacco and Health : The Indian Scene. L.D. Sanghvi and Perin Notani (Eds.)
UICC Workshop, Tata Memorial Centre, Bombay 400012, India. (1989)

An Intervention Study to Control and Contain Oral Cancer

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In a house to house screening survey, 36,000 tobacco chewers and smokers were selected from the rural population in 3 districts of 3 states in India namely Kerala, Andhra and Gujarat. These individuals were interviewed for their tobacco habits and examined for the presence of precancerous lesions in a baseline survey and then annually over the last 9 years. They were educated using personal communication as well as mass media approach to give up their tobacco habits. The results have indicated consistently that it is possible to induce changes in the tobacco habits of rural populations through educational efforts. This is further substantiated by a significant decrease in the incidence of precancerous lesions. As most cancers in India are reported to be preceded by precancer, this study demonstrates that the primary prevention of oral cancer is feasible, effective and practicable.

Introduction

India is inflicted with the problem of oral cancer. Here the incidence of oral cancer is higher than the incidence of most other cancers (1). The relationship between oral cancer and tobacco habits is indeed established as much beyond doubt as the relationship between cigarette smoking and lung cancer though it may not be as much publicised as the latter. Since such is the state of affairs, it appears logical that one way in which oral cancer can be controlled is by intervening in the tobacco habits of the people. The disease can be arrested and cured by treatment but the epidemic would be impossible to control if the tobacco habits are not checked. As in the case of lung cancer, in oral cancer too, it is yet not known how much usage of tobacco would actually result in oral cancer. It seems improbable that such a guideline would be available in the near future. For the human personality, tolerance to disease, threshold of vulnerability might play an important part. Therefore it would appear that our best bet would be to make people leave the tobacco habit. Thus the primary health care delivery system ought to include such

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education.

The question then arises : how to design such a system ? Not all deliveries of health care meet with success. Unless a health care delivery system is designed keeping in mind the target population, their habits, beliefs, lifestyles and knowledge, the system is likely to backfire.

The Tata Institute of Fundamental Research has made a specific attempt at designing a delivery of health services to make people leave their tobacco habits. The communication strategies utilized are those designed and developed with the full involvement of target population. The project study went through two phases before it undertook to intervene in the tobacco habits of the people. Phase I was a series of cross-sectional field surveys undertaken with the objective to determine the prevalence rates of oral precancerous lesions and their association with tobacco habits. The total population surveyed was over 155,000 (2–4). Phase II of the project was a ten-year follow-up study of 30,000 of the original study population. The results obtained in phase I and II established that a) oral cancer and precancerous lesions occurred almost solely among those who smoked or chewed tobacco and b) oral cancer was almost always preceded by some kind of precancerous lesions (5).

On the basis of these results intervention study was launched. The objective of the study was to make people leave the tobacco habits and to investigate any effect this might have on incidence and regression rate of precancerous lesions and in thus controlling oral cancer.

In the present study, the intervention efforts have been implemented through a carefully designed comprehensive programme in a specifically delineated target population. Every individual in the target population is being examined annually to measure the changes in the incidence and regression pattern of the disease. The intervention programme thus is carried out by a team of professional dental surgeons and social scientists who are given special training in conducting in-depth interviews and in approaching and interacting with the habitues (i.e. those who use tobacco) in a considerate, sympathetic, warm and positive manner without being condescending in any way.

Materials and methods

Study population : The study population was selected from Ernakulam district in the state of Kerala, Bhavnagar district in the state of Gujarat and Srikakulam district in the state of Andhra Pradesh. A large number of individuals were screened and about 12,000 tobacco habitues aged 15 years and over, were selected in each district. The tobacco habits practised in the study included smoking cigarettes, bidis, smoking chutta in conventional and reverse manner, and chewing tobacco with or without betel quid.

Intervention methods : A program of intervention was chalked out after appropriate pilot and pretesting surveys. In-depth interviews of the habitues were conducted to investigate the reasons for starting and continuing the tobacco

habits, the perceived implications of the habits in social, economic and health aspects and the possible reasons that could make them give up their habits. Also pilot surveys were conducted to assess the communication media facilities available to the target population which could be employed for conveying the intervention message. The intervention program outlined a timetable for employing different media of communication and regulating the flow of information so as not to overwhelm the target population. Pilot studies also helped in modifying the messages whenever necessary, with a view to making them easily understandable. All this was an on-going process for every follow-up.

This paper comprises the results upto 8th follow-up. Each follow-up had utilized strategies of communicating messages to the target population which assisted and enabled them to give up the tobacco habit. The unique feature of this project has been its user based philosophy which is characterized by providing appropriate input depending upon the stage of the subject as follows :

1. Knowledge regarding association of tobacco habits and oral cancer was imparted.
2. This knowledge was further strengthened by visual reinforcement through flip charts, posters, films, etc. and a little amount of fear was deliberately induced.
3. Knowledge of health and other benefits on quitting the habit were conveyed : regression of lesions, saving money etc.
4. Various possible methods of discontinuing of tobacco habits such as cold turkey, gradual reduction, postponing the first smoke or quid, etc. were described and the most appropriate one for the subject was suggested depending upon the psychological profile of the subject and characteristics of the habit.
5. Withdrawal symptoms were explained and emphasis placed on their temporary nature.
6. Appropriate praise and reinforcement was given and leadership role suggested for successful quitters.

The modes of communication utilized were as follows :

1. Personal communication : This indicated one to one contact with the target population. This was done with a view to helping them sort out doubts and learn behaviours which would result in abstaining from the habit. The steps in personal communication were worked out in a logical order keeping in mind the dynamics of human psychology and what it takes for people to make decisions and change their attitudes and behaviour. Certain visual aids were utilized in carrying out the personal communication.

2. Films : Two films were produced specifically for this study.The objective of the first one was to give information and create awareness on the relationship between tobacco habits and oral cancer. The objective of the second film was to point out how habits are taken up and developed. In addition how they can be given up. Film is a very powerful medium of creating awareness and when effectively utilised works in motivating behavioural changes.

3. Posters : Posters were displayed with an objective to serve as a reminder to the target population that they ought to be reconsidering their tobacco habits. Two kinds of posters were utilised, one with written messages only and the other with a visual display along with the written message.Slides were prepared from those posters and were projected in movie houses in the vicinity to serve the same objective. In addition, hand written posters made by summarising the finding of the particular village,were left behind to act as a reminder that they have been examined for oral cancer.

4. Newspaper articles : These were published to inform and educate people on the subject of oral cancer. It was known that many members of the target population do not read, nevertheless these could be read by some including schoolgoing children who are found to be important agents of change in rural India.

5. Folk drama : These were enacted with an objective to talk about the prevalent superstitions and misconception about tobacco.

6. Radio programs : These were designed to motivate people to give up their tobacco habits in light of the fact that these might lead to oral cancer. These were utilised to create an environment to help the target population to think about their tobacco habits.

7. Cessation camps : In the last two follow-ups we have been holding cessation camps at their door steps. This was being done to address the demand of our target population who told us that they did believe tobacco was bad and would like to give it up. They wanted to know how to do it. In cessation camps detailed discussions were held to suggest solutions to the problems in discontinuing the tobacco habits. Regular daily follow-up was maintained for a few days.

8. Others : In addition to this,clinics were run to address their dental complaints thereby trying to eliminate at least some of the reasons given by them for practising the habit. Also exhibits and group meetings were arranged to show the common lesions and hold discussions that might help the target population to ask questions and give vent to their feelings and doubts.

These inputs were assessed individually through carefully designed questionnaire-based sample studies for their impact as per their objectives. Since they all impinge on the same human being, it is hard to ascertain which actually led to the decision. A rank ordering by the target population of these inputs as per their preference revealed that they have been helped the most by one to one interaction; and cessation camps had also aided them immensely.

Results

Results have consistently been in the positive direction (Table 1). Considering that the target population was not naturally motivated to give up the habit it is quite remarkable that such results were obtained.

Table 1. The stoppage of tobacco use in successive follow-ups.

Follow-up	Bhavnagar	Ernakulam	Srikakulam
	%	%	%
First	4.1	2.0	4.9
Second	7.6	4.4	10.9
Third	10.5	6.4	14.2
Fourth	11.6	7.6	15.4
Fifth	12.5	9.4	16.6
Sixth	14.1	11.2	17.0
Seventh	11.5	11.6	17.3
Eighth	12.2	12.3	17.4

Discussion

A detailed assessment of the project was made after 1 year and after 5 years. After 1 year it was found that the regression of lesions was significantly higher among those who had stopped or reduced their tobacco use than among individuals who did not reduce or stop their tobacco use (6). After 5 years it was found that a significantly higher percentage of individuals had stopped and reduced their tobacco use compared to the control cohort. As a consequence of this increased stoppage and reduction in tobacco use, the incidence of oral precancerous lesions decreased significantly and substantially in two out of three study areas (7). Through a multiple logistic regression analysis it was shown that the intervention efforts definitely helped people in stopping their tobacco habits (8). Further results show that either individuals are continuing with abstinence or if there is a relapse among some individuals it is more than compensated by other individuals giving up the tobacco use.

Where do we go from here ? How can this project be used as a take off point for intervention at the national level ?

We have examined the feasibility of training the basic health workers to examine the mouths for early detection of lesions both precancerous and cancerous and we have found this to be possible (9). It would be indeed worth while to assess the possibility of training these health personnel to intervene in the tobacco habits. If it is possible, the potentials are great. Most of the strategies of communication that are utilised effectively in this project are those that involve mass media or an approach that can be used on a large scale. Undoubtedly this needs to be validated.

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Intervention Studies of Tobacco Behaviour Modification in Kerala State

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The Community Oncology Division of the Regional Cancer Centre was started in May 1985 and since then is engaged in various intervention programmes in Kerala state, to modify tobacco related behaviour. These programmes are discussed in the paper.

Introduction

Kerala is a small state, but it sells the maximum number of cigarettes among all the Indian States. It also has the maximum number of registered bidi workers' co-operative societies. In addition to this, there are a sizeable number of independent bidi makers. According to official sources, about a lakh of foreign cigarettes flow into the state every day. Though little tobacco is cultivated here, Kerala is one of the major consumers of chewing tobacco. As a result of all these factors the load of tobacco related diseases would be high in the state. Tobacco related cancers account for more than 50% of the male cancers in Kerala. Cancer of the oral cavity is the second commonest cancer site in women; accounting for nearly 19% of the cases. If one observes carefully, it can be seen that the mean age of lung cancer in Kerala is about 10–15 years younger than that of lung cancer in developed countries. Lung cancer is the commonest cancer in man, in the most productive age. This reduction of age is seen for the pattern of coronary heart disease as well in the state. All these factors motivated the Community Oncology Division of the Regional Cancer Centre to take up a few tobacco intervention programmes in Kerala.

A glimpse into history shows that Cochin, one of the erstwhile states of pre-independent Kerala, can be credited to be the pioneer in making legislation restricting the use of tobacco, as early as 1920. In the act made by the Maharaja of Cochin, the sale of tobacco was forbidden to minors. There were even provisions to punish the minors, if tobacco was found on them by the school teachers or policemen. I may also point out that a major office in Kerala, the Cochin Port Trust has completely banned smoking within its premises recently, and the ban is rigidly enforced.

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The anti-tobacco activities of the state will be discussed in detail.

Activities of college students

The anti-tobacco activities aimed at the college students are executed through the National Service Scheme (N.S.S.) volunteers. This project is discussed in greater detail in a paper presented in another session. Over 300 units of the N.S.S. participated in the activities during the current academic year. There were 5 one-day workshops for the programme officers and selected volunteers of all the colleges in the months of July and September 1986. A poster and slogan competition was held. The first phase of activity was marked by a public meeting held in each of the colleges on the birth anniversary of Mahatma Gandhi i.e. 2nd October 1986, to explain the harmful effects of tobacco. School teachers, local medical practitioners, and representatives of social organisations were invited for this meeting. At the end of the meeting a pledge was taken by the N.S.S. volunteers to keep away from tobacco, alcohol and drugs.

The second phase of the anti-tobacco activities was undertaken by selected colleges of the University of Kerala and the University of Calicut which had health education as a major item for their 10-day camps in the adopted villages. Public meetings were organised in the villages in the late evenings and the students and other resource persons spoke to the villagers on the harmful effects of tobacco, the economics of smoking and chewing, advantages of quitting the habit etc. Local school teachers, grassroot level workers of the Health, Community Development, and Agriculture Departments, and members of local youth clubs were specially invited for these meetings. Some health education material was printed and distributed to these villagers by the students. Till 31st March 1987, 20 colleges of the University of Kerala and 16 of the University of Calicut have completed the second phase of the activity.

Activities of school children

The activities targeted for school children are to be launched in the next academic year. A trial to determine the feasibility of this programme was carried out in January, 1987 at Christ Nagar English High School, Trivandrum. A combined meeting of the students and the members of the Parents-Teachers Association was held in which they were explained the "Ifs and Buts" of smoking, as given in the booklet prepared by us. An oath was taken by the students and willing parents to abstain from tobacco, alcohol and drugs. Each student was given active participation in the anti-tobacco campaign by making him sell three copies of the book at a rate of Rs. 4.00 per book. Students who sold more than 12 books were presented with T-shirts printed with the anti-tobacco slogan. The first attempt was very successful and 9,000 copies of the book have been sold so far.

Activities of health personnel

The Regional Cancer Centre, Trivandrum, is planning to train multipurpose

health workers of half of the District of Trivandrum to study the feasibility of using trained health workers in the early detection of oral cancer and pre-cancerous lesions and in delivering health education regarding the harmful effects of tobacco and in particular its association with oral cancer. These health workers while trying to create awareness in the villagers about the harmful effects, will also emphasize the "bad" economics of tobacco usage in a person to person communication method. They will impart health education to organised groups like *Mahilamandals* (Women's groups) and youth clubs of the locality. Provision will be made to evaluate the fluctuations in the tobacco habits of the study area versus control area.

Activities of voluntary and social organizations

Voluntary agencies and social organisations are involved in a big way in the cancer control and prevention activities in the state. Cancer awareness classes are given to various organisations in which they are explained the harmful effects of tobacco and its role in the production of cancer and other illnesses.

On occasions when a class could not be conducted a video tape was circulated to organisations with the facility to screen such audio-visual talk. Public education talks were given in cancer detection camps too.

Activities through communication media

The communication media was also actively utilised to influence the tobacco behaviour of the target group. Five articles were published in the lay press about tobacco related cancer, hazards of smoking, etc. Talks and discussions were broadcasted on the radio. A programme about the Regional Cancer Centre was televised in which information about tobacco related cancers was given. Slogans against tobacco and the warning signs of cancer were aired between programmes on the stations of All India Radio. Warning slides on ill effects of tobacco usage were projected between programmes on television.

Several other activities have been started in the state. A new organisation called "Pukavali Nirulsahana Samithi"–forum for discouraging smoking has been started since 1st October 1986, with its headquarters at Trichur. A clinic to help smokers to quit smoking is being opened under the joint auspices of the Department of Psychiatry, the Department of Sociology and the Department of Community Oncology of the Regional Cancer Centre.

This paper has covered in brief, the highlights of the intervention programmes which are being planned or already underway in the state, to modify tobacco behaviour of the people. Needless to say proper planning, continued support and guidance from international and national agencies are essential to carry on with these activities to counter organised propaganda of tobacco companies.

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Development of Intervention Studies in Karnataka to Modify Tobacco Related Behaviour

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A survey-cum-education project in rural Karanataka was envisaged to assess the efficacy of an anti-tobacco community education program. The objectives are : 1) to prevent onset of tobacco habit in the non-habitues and 2) to stop the habit in the habitues. The paper describes in detail the first phase of the project.

Introduction

Tobacco was introduced in India in the sixteenth century. However, health hazards of tobacco use came to be realized a few decades ago. In the urban and rural area, chewing of betel quid has been a traditional custom. But, the incorporation of tobacco into betel quid and smoking of tobacco as bidis and cigarettes lead to a tenacious habit which over generations has become a problem of human behaviour. Furthermore, India has become a target for aggressive promotional campaigns, especially for cigarette consumption, by transnational companies (1). It is now feared that the known and potential health hazards resulting from the use of tobacco will make it one of the major health problems in India (2). Based on crude incidence rate of the Population Based Cancer Registry (Bangalore) in Karnataka, the estimated number of new cases of cancers of all sites was 28,336 in 1986 (3). This number is likely to increase to 32,138 in 1991 and 38,209 in 2000 AD. Tobacco related cancers accounted for 7,161 cases in 1986. The number is likely to be 8,140 in 1991 and 9,680 in 2000 AD. Tobacco related cancers constitute 25–30% of all cancers. The problem of tobacco use is thus gigantic and calls for a concerted socio-scientific action to reverse the escalating trend.

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At present, data on tobacco chewing and smoking patterns in the population are meagre. The data on age and sex-specific prevalence and incidence rates and specific disease pattern vis-a-vis the type and mode of use of tobacco are also meagre. The knowledge related to these facts is prerequisite for devising the strategy to modify tobacco related behaviour in the community. The strategy must necessarily be multi-faceted, because the problem is not only one of human behaviour but also economic since the existence of small farmers and the large number of people engaged in the processing, manufacture and sale of tobacco and tobacco products is linked with it (4).

Is it feasible to modify human behaviour? If so, what are the strategies? One of the strategies is to mount intervention studies to modify tobacco related behaviour. Human behaviour is very complex to unravel. It becomes confounded if it is habit related as in chewing and/or smoking tobacco.

Project

The survey-training-education project, spanning the disciplines of public health and social sciences and utilising the services of staff specially recruited for the project and of the staff within the health infrastructure in rural Karnataka has been launched in Kolar District of Karnataka.

The following are the project objectives :

Main

1. To prevent onset of tobacco habit in the non-habitues in the community.
2. To stop the tobacco habit in the habitues (i.e. those who use tobacco).

Subsidiary

1. To train Primary Health Centre personnel to provide health education to modify tobacco-related behaviour in the community.
2. To train Primary Health Centre personnel to inspect the oral cavity, identify lesions and classify them into referrable and non-referrable categories.
3. To design, pretest, produce and utilise suitable health educational material for modification of tobacco related behaviour and consequently prevent oral cancer in the community.

The project is planned to be conducted in three consecutive phases. The first phase involves making general preparations and training of the staff for field survey. The second phase is to undertake intervention study and the third phase is to evaluate the impact of the intervention study.

A preliminary study of prevalence of tobacco related habit was conducted in Kolar District. The study showed that tobacco chewing habit ranged from 13% in people of Malur to 68% in people of Dibbur. The prevalence of tobacco chewing among 41,711 persons above the age of 5 years living in Chickballapur Taluka was 40.3%. 'Kaddipudi' powdered tobacco plant stem, was the most commonly used form (83.4%) (5). This habit is peculiar to this region especially in women (Fig. 1).

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Fig. 1. Woman making the quid

1. Tobacco stem
2. Kaddipudi (powdered tobacco stem)

Phase I

Preparation

1. Preliminary discussions were conducted with the key personnel of the Health and Family Welfare Department to involve the existing health infrastructure in the project. The District Health and Family Welfare Officer of Kolar and all his staff conducted preliminary sample survey to ascertain the tobacco related behaviour among people of clusters of villages in Kolar District. Demographic and other data of Dibbur (experimental area), Malur and Gudibanda (control areas) Primary Health Centre areas, were collected (Map-1).
2. Project personnel were recruited and trained in principles of community health research and interviewing.
3. Schedules were drafted and preliminary testing was done through interview of patients and family members at the Dharmashala of the Institute.

Survey

The baseline survey in the experimental and control areas was planned with

MAP 1

ICMR & KIMIO ASSESSMENT OF THE EFFICACY OF AN ANTI-TOBACCO COMMUNITY EDUCATION PROGRAMME

Map of Experimental Area and Control Areas in Kolar District

four inter-related methods of data collections :

1. Village appraisal
2. Household survey
3. Individual interview with household members having tobacco related habits.
4. Screening of household members for tobacco related health problems.

For the regular survey, villages were identified in Dibbur experimental area and in Malur and Gudibanda control areas so that approximately 10,000 population was available in each of the three areas.

Pilot study in Begali village

Pretesting of the four schedules in a spot survey of Begali village was done. Begali is 5 kms out of Kolar and has a population of 1,430 (743 males and 687 females). Eleven social investigators and one dentist took 5 days for the survey. This survey helped in the training of the project personnel to develop necessary skills through field-team-personnel interactions.

Village appraisal was carried out by social investigators through group discussions with village officials and local leaders of each village. This effort was for collection of data on socio-economic features and community leadership pattern. Identification of possible volunteers to assist in the project activities was also done. Notional map of the village and numbering of households was also undertaken (Map-2).

Household survey consisted of collection of information on economic status and on members who chew and/or smoke tobacco. This information would assist in the Phase II and III of the study. Interview of habitues covered questions on knowledge, attitude and practice.

Screening of the habitues above 15 years of age for tobacco related lesions/ diseases was another activity.

The salient findings of the pilot study (6) were :

1. 48.3% were tobacco habitues.
2. Female habitues predominated (53.1%).
3. The tobacco habit started in 11 – 20 year age group in 71.2% of habitues.
4. There were three habitues below 15 years of age.
5. Most of the habitues (73.4%) were illiterate farmers-cultivators and agricultural labourers.

6. Tobacco chewing was prevalent in 63.2% of the habitues, and chewing habit was seen in twice as many women as men. The type of tobacco used was 'Kaddipudi' by 89.5%, tobacco leaf by 10.2% and both by only 0.3% of chewers.
7. Among the 152 tobacco smokers, the majority (88.2%) were bidi smokers, 5.9% cigarette smokers and the rest used bidi and cigarette. There were no female smokers.
8. The habitues spent nearly 1/4th of their income on tobacco either for chewing or for smoking.

Observations of tobacco related behaviour among the chewers indicated that 91.8% of chewers felt that they happen to get into the habit and then continued. A small proportion of habitues mentioned that they took to tobacco chewing to prevent bad breath or to overcome toothache.

The need to relieve tension and to have relaxation was the dominant compulsion among a large proportion of smokers. Smoking is a social habit and for many, smoking also provides pleasure and strength. There is a very thin line between habit and addiction. Eighteen percent of smokers had been advised to stop and 10% tried to stop smoking. Among the habitues 12% felt that tobacco use led to domestic, economic and health problems. Only 1% of habitues were aware of anti-smoking messages. Twenty eight percent also felt that tobacco use was harmful and not respectable.

Clinical examination showed that 1/6th of the people of Begali had respiratory tract problems such as chronic cough (13.6%), cough with phlegm in the morning (16.7%) and sore throat (19.7%) and 20.2% spoke of having bad breath. Oral cavity abnormalities observed were stomatitis (9.4%), dry mouth (22.5%), increased salivation (14.7%), trismus (2.8%), periodontitis (69.5%) and gingivitis (30.5%). Among the 213 people examined clinically, there were 22 cases of leukoplakia (18 females and 4 males) and 2 cases of nictotina palati. The prevalence of leukoplakia was 10.3%.

Training

The training of the health personnel is a sequential process with focus on :

1. Identification of the training needs.
2. Utilization of the key personnel of Primary Health Centres.
3. Action oriented job responsibility–category-wise and activity-wise.
4. Mobilization of faculty.

The strategy adopted in the training should be of practical utility with a thrust on programme objectives. The training objective should be such as to minimise the gaps between research methodology, training methods and field practice. Systematic training, evaluation and field performance would assist in mid-course correction. Training and retraining is a continuum. District level managers and Primary Health Centre level supervisors constitute a link between the project team and the health assistants.

The training program needs to be planned so that the Primary Health Centre staff, in addition to their routine work load would be motivated to undertake the extra burden of intervention study thus leading to cooperation in implementation of the same in the field. In the training sessions, the programs and educational objectives also need to be stressed.

The problem for the trainee is how best he may maximise motivation in the target population. The trainer, therefore, has to impress on the Primary Health Centre staff their role in the community. Clear understanding of the skills and aids is prerequisite for reaching effectively the target audience.

Briefly, training of key personnel in a phased manner is essential but this should be achieved without prejudice to the ongoing activities. The sustained efforts by the network of Primary Health Centre staff will, it is hoped, change the tobacco related behaviour in the beneficiary.

The first component in the module for training of the Primary Health Centre staff is with regard to the tobacco related behavioural change, while the second is an illustrated manual providing knowledge on anatomy, etiology, precancer, cancer and procedure for clinical examination.

Preparations of health educational material

It is not easy to counter the sales-promotional approach of tobacco industry, which is not based on product information, but on enhancement of individual's self image and boosting of the ego, combined with all kinds of pleasurable and desirable concepts. Hence a simple educational approach, which may even arouse fear and is bereft of any entertainment value may not have the desired effect on the target group. To meet the programme objective-1 i.e. prevention of onset of tobacco habit in non-habitues, the first requirement is to determine factors which lead to tobacco related behaviour. Subsequently, educational material, keeping in view the above considerations, would be prepared to impart knowledge regarding :

a) The ill effects of tobacco on health and in particular the association of tobacco habits with oral, pharyngeal and lung cancers.

b) The benefits of not smoking and chewing.

For meeting programme objective-2, which is to achieve stoppage of tobacco habit in the habitues, in addition to 'a' and 'b' above, the programme would also emphasise

c) The economic loss due to tobacco use and thus help develop favourable attitude to stop tobacco habit.

For each of the educational objectives, suitable methodology, media and messages will be devised.

Media : The media has an important role to play in public information programme which is a mass communication approach aimed primarily at creating and intensifying awareness of the public, regarding the problems of tobacco usage.

These programmes would :

a) Sensitize, provoke tobacco habitues with the message that tobacco consumption causes oral and other cancers in addition to other diseases of respiratory and cardiovascular systems.

b) Discourage chewing and smoking in the non-habitues.

c) Educate the population regarding health risks and socio-economic implications of chewing and smoking.

Such programmes are expected to create an atmosphere conducive for producing desired results when education programmes, on a person-to-person basis, will be carried out in the target group.

Since certain forms of tobacco use and chewing of betel are habits developed by custom and tradition of the region, approaches in health education will be devised which would be relevant for local situation. A package of media consisting of folders, flash cards and exhibition sets, hand bills, slide cassette program and uniconcept film on chewing and smoking will be developed.

All materials, media and strategies will be pretested and utilized as appropriate for the various target groups such as health personnel and the experimental communities in various categories of age, sex, habit patterns of tobacco use, socio-economic millieu and other factors native to the community/village.

Evaluation points and strategies will be inbuilt aspects of the health education component.

Phase II

The second phase is the phase of intervention. This involves the following activities :

1. Active intervention through intensive health education by the Primary Health Centre personnel in the experimental area.
2. Offer of appropriate services to persons seeking assistance of the Primary Health Centre personnel through referral to appropriate level of health/medical care centre.
3. Undertaking annual repeat surveys for tobacco usage prevalence.

Phase III

The third phase of the study will be utilized for determining :

1. The overall impact of health education programme through existing health personnel on tobacco usage practices in experimental population vis-a-vis the control population.
2. The effect of the programme on the incidence, prevalence and regression rate of oral lesions in persons above the age of 15 years, with the help of dentists.

It is hoped that intervention studies using health education materials

developed and delivered through the health infrastructure will significantly modify the tobacco related behaviour.

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Health Education in Children

Anne Charlton[1]

Introduction

The problem of tobacco usage by children has its primary roots deep in the past. Tobacco has been a part of life in families throughout the ages; chewed to relieve hunger; chewed to relieve pain; chewed in pan; chewed in quid; smoked in hookah, chutta, bidi, cigars, pipes and cigarettes. All these methods of tobacco usage developed long before the harmfulness of tobacco was suspected. Now its harm is known and proven beyond all shadow of doubt, and it is necessary to break the cycle of acceptance which has become so deeply entrenched in people's lives. To prevent children from starting to use tobacco we have to combat both the established acceptance by the family and the community and the blandishments of the tobacco industry. Education is one of the most important elements in this process.

Identifying the problem

Before any intervention can be undertaken, the problem must be identified. In this way planning can take into account the local situation. It also provides a baseline against which the effectiveness of any method which is tried can be evaluated.

Need No. 1 : To develop a standardised questionnaire or questions and standardised methods of administration so that the comparative rates of tobacco usage in widely differing communities can be measured. The simplicity or the sophistication of this measurement tool will of course, depend on the level of education in the areas concerned, but it is always best to avoid long complicated questions and questionnaires. What really needs to be known is the prevalence of tobacco usage in that particular locality, who uses it, and why they use it.

It is at present impossible to compare rates of tobacco smoking and chewing between children in various countries of the world because of variations between the surveys which have been carried out. Most have concentrated on cigarette

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smoking. For example surveys in recent years have shown prevalence of regular smoking at the age of 14 years to range from 10% in Argentina to 26% in Denmark for boys and from 5% in Argentina to 35% in Denmark for girls (1, 2). However, it is essential that these figures should not be regarded as comparable for the reasons mentioned earlier.

It is also vital to repeat surveys at regular intervals. The problems of leaving a long gap between surveys, and of surveying only a particular group has been demonstrated in England; where a gap of 16 years existed between the national survey of boys only carried out in 1966 (3) and the next national survey of boys and girls carried out in 1982 (4). It meant that for the whole of these 16 years the findings of the 1966 survey formed the basis of all thinking and planning related to children and smoking. But by 1982, the 1966 children were already the parental generation, lifestyles had changed, and there was no baseline against which to measure girls' smoking.

The general tendency throughout most countries of the world at present is for girls' smoking to be increasing, whilst over the last twenty year period, boys' smoking has decreased (5).

There is often little change overall in the short-term even where intensive anti-smoking education programmes are in action (6), but in the long-term almost all westernised countries have shown some changes. For example, in Sweden, where education authorities have made a sustained effort to educate about the ill effects of smoking and other legislative measures have been instituted, cigarette-smoking prevalence fell from 41% (boys) and 47% (girls) in 1971 to 21% (boys) and 33% (girls) at age 16 in 1980 (7).

However, this is not the case in all countries. In Africa, South American countries, and probably in India, cigarette smoking is thought to be on the increase among young people. The rise could be due to the action of the tobacco industry which, finding their sales to be decreasing in countries where cigarette smoking used to be popular but has declined, are trying to boost their sales by intensive marketing techniques in countries where cigarette sales were minimal.

The micro-climate

There are several aspects of micro-climate which influence a child's tobacco usage, and these are discussed below.

The home and family

The first people a baby sees and recognises are its parents and other members of the household. Young children's first drawings show mother as a big face – a predominant object in baby's life – if it always has tobacco of some form in it, it becomes part of the baby's image of this most important adult.

Parents' smoking or tobacco usage affects children in several ways.

(a) It has been shown that children whose parents smoke are almost twice as

likely to be smokers as those whose parents do not smoke (8). There is probably as yet little hard evidence for a similar effect for the smoking of bidi or chewing pan or quid, but it might be guessed that it would be similar.

It is mother who gives her child some tobacco to chew to appease his hunger or ease his toothache in the fields. It is mother who gives some pan to chew at the end of the meal. It is mother who leaves her cigarette burning on a low table where the small child can try a puff of it. Whatever is done to prevent tobacco usage among children must involve mother, father and the extended family as well as the local communities in general.

(b) There is evidence that some children try a cigarette before the age of five years in England (8). The age of 9 years for boys and 10 years for girls is the peak for experimenting with a first cigarette. A survey showed 11·7 years as being the peak age for Indian boys (1) to try a first cigarette. However, tobacco chewing and smoking of bidi could, and do, begin much earlier. The availability of tobacco materials in the house makes it easy for a child to have access to these.

(c) Parents' cigarette smoking certainly increases the risk of children to develop certain illnesses. Coughs, asthma, pneumonia, middle ear complaints and possibly the long term risks of lung cancer are increased in children whose parents smoke (9).

(d) Most importantly in the case of cigarette smoking, whether or not parents are smokers, evidence has shown that if they express strong disapproval of their children's smoking, the children are seven to eight times less likely to be smokers than if their parents approve (10, 11). This may be the same for chewing tobacco or smoking bidi, but first it is important for the parents to recognise the problem and the necessity for them to influence their children's decision on tobacco usage.

Need No. 2: To develop means of convincing parents of their important role in preventing their children from taking up the tobacco habit. A way of making them aware of what they can do e.g. not use tobacco themselves; or if they must use tobacco, especially when smoked, to do so away from their children and also perceptibly disapprove of their children's tobacco usage. It may be necessary to take the parental and community education much wider than the tobacco issue and include e.g. education on treating toothache, or on how to have better and cheap snacks in the fields and so on.

Friends

Friends are soon met, even before the child leaves home, but certainly when he or she starts school. Much research has shown that children are vastly more likely to smoke if their best friends do so and this is likely to be so with other methods of tobacco usage. Best friend's opinion can carry most weight with the smokers. For example in one study, only 2% of the children smoked if they thought their best friend would not approve (11).

Peer pressure to use tobacco is strong. Children want to be one of a group and find it very hard to refuse tobacco if their own group of best friends use it. Numerous studies in western countries have consistently identified an "at risk" group for cigarette smoking (12). The children most likely to take up cigarette-smoking have a distinct set of characteristics i.e. they are often low-achievers at school; they are bored or "fed-up" with school; they want to grow up quickly and be free of confines of school; they often have a special best friend or friends who are older–over school age; sometimes but not always they are rebellious; they drink alcohol more often; they go to discos and rock concerts more often. Users of bidi, pan or quid might have other characteristics, but it is probable that the young cigarette smokers in India might share many of the characteristics of "at risk" groups in other countries.

Need no. 3: To know the characteristics of the children most at risk in India.

Need no. 4 : To find ways to help these children with their problems. Group work, new interests in school etc., and also lessons to help them find ways of saying "no" when friends encourage them to use tobacco, need to be developed.

Teachers

There is evidence that school teachers can be influential in children's decisions to smoke or not to smoke. Their influence has three main strands : the personal example of the teacher; development and implementation of a school policy on tobacco usage; teaching intervention lessons.

A very few popular teachers might act as role models, but any teacher who uses tobacco legitimises the habit for the children because a teacher is seen as a knowledgeable authority figure (13). Research in various countries has shown for example, that in schools where fewer teachers smoke, fewer children are smokers (14). On the whole, in most countries now the level of cigarette smoking among teachers tends to be lower than for the general population e.g. 17% of men teachers and 19% of women teachers in England smoke as opposed to 36% and 32% nationally (15). There was however, a considerable amount of "hidden" smoking (14%) of pipes and cigars among men teachers. Perhaps tobacco chewing is comparable among Indian teachers.

All schools need some definite policy on tobacco usage which applies to both pupils and teachers. There is research evidence that pupils who leave stricter schools are less likely to be smokers in the future (16).

Before teachers would be willing to accept a policy on tobacco usage for themselves, it is necessary that they appreciate the health risks and that they are made fully aware of their possible influence on young children with regard to the tobacco habit. In a Cancer Research Campaign-funded survey in England, 85% of nonsmoking teachers and exactly the same percentage of smoking teachers would reprimand a child from their class for smoking in school (17). It was as parents

that they differed; 83% of nonsmoking teachers and 71% of smoking teachers being willing to reprimand their own child. In many countries now, it is a rule that no teachers or pupils are allowed to smoke on school premises or on school outings and outside activities related to school (1). It can be difficult to attain this ideal situation because of teachers' opinions. In the same survey, only 4% of the smoking teachers felt that teachers should not be allowed to smoke in school (17).

If there is a school policy, it is vital that all the teachers and pupils know it, otherwise it is impossible to implement it (18).

Need no. 5: To develop a clear policy with regard to tobacco use in and in association with schools, for both teachers and pupils.

Teachers can also be central in teaching tobacco intervention, both as lessons to children in school, and by taking a wider role in initiating and developing community and family-related projects. If it is the norm for the family to smoke or to chew tobacco, it is unlikely that the school alone will have much influence simply by teaching lessons to the children (19, 20). All the significant background people need to be involved.

In many western countries there are now many excellent teaching packages for intervention against cigarette smoking specifically (12). In Western Australia, a very ambitious and successful programme involved schools, the media, doctors, the whole population in fact (21). It started with an exciting launch involving competitions and much publicity. It was a very important initiative, and could form a good model for countries similar to Australia. A most important proviso, however, is that it is never possible simply to transfer any programme from one country to another. Basic principles can sometimes be transferred, but local cultures are too different for programmes suitable in one area to suit another.

The basic premise with regard to school and community programmes appears to be to cover all aspects of the tobacco problem. One of the most successful programmes was developed and evaluated in Norway (22). Not only did it involve families as well as the schoolchildren, but it included informational aspects, pupil involvement and social aspects. The Health Education Authority in England has taken on this programme and is adapting it for British use – but with considerable difficulty due to the Norwegian basis and purpose of the materials.

Health risks information alone is boring, too far ahead in time to be relevant to children, possibly frightening especially if the child's parents use tobacco, and it can also encourage the children to use tobacco because they like to take risks ! But children do need to understand the health risks.

Social aspects can involve stained teeth and fingers, smelly clothes and hair, saving money, and alternatives to using tobacco e.g. diets to control weight, real ways to prevent and cure toothache, cheap light snacks for taking to work etc. Added to the health risks, these aspects can help children to see reasons for not using tobacco.

But one of the most effective approaches of all, has been developed in America (23) and in some other countries : it helps children say "no" when they are offered a cigarette. Once again the actual materials could not be transferred, but the principle could be.

Three crucial phases in the history of children's cigarette smoking have been identified, (a) at 9-years-of-age when many of them experiment with their first cigarette, (b) at 13-years-of-age when they become regular smokers, (c) at 16-years-of-age when they want to stop smoking. The Brigantia Smoking Prevention Programme targets on these age groups and is proving to be very successful (24). The crucial phases of the various kinds of tobacco use in India may still need to be identified, but it is clear that timing of the intervention is almost as important as the method used.

To sum up, with regard to teachers' involvement

Need no. 6: To undertake surveys of teachers' tobacco usage and their importance as role models.

Need no. 7: A definite recommended policy on tobacco usage for all schools, including teachers and pupils is required. A Governmental mandate makes the enforcement and acceptance of the ruling smoother, rather than if there are individual policies made by individual headteachers.

Need no. 8: To develop good relevant teaching materials for schools, but more importantly still a community and family-based programme to involve everyone. (The Brigantia Smoking-Prevention Programme for 9-year-olds which does involve the family has brought about a significant drop in the number of fathers smoking, as well as deterring the children from taking it up) (24).

The macro-climate

Whatever is done on the individual and family scale to prevent children from using tobacco, it can only be set in the wider context of the national and regional climate. Children's tobacco usage is to some extent subject to the influences of the people and circumstances in each child's own microcosm, but these microcosms do not exist in isolation. The comparative strengths of the various macro-elements varies from country to country, but they fall basically into three main forces

(a) the national acceptability of tobacco as a crop and as an industry

(b) the availability of tobacco to children

(c) the advertising.

Alternative crop

Children together with their families are involved in the cultivation of the

tobacco crop in India and with the home industry of bidi-making. To take away the tobacco crop without replacing it with some other equally, cost effective crop would severely effect the livelihood of many people.

Need no. 9: To identify suitable food crops to replace the tobacco, thus providing income, employment and food.

Legislation

Tobacco is available to children in the fields and in the home in many parts of India from the time the children are infants. In other countries, where cigarettes are sold, many children try their first cigarette before they are five years old, especially if mother smokes. Many countries have developed legislation to help prevent the sale of tobacco to minors (usually under 16 years of age) (1). Australia, New Zealand, U.S.A., U.K., numerous countries in Europe, Japan (where the limit is under 20 years) and certain part of South Africa, have such legislation. Sometimes there is a clause which makes an exception for tobacco products that the vendor has no reason to believe are for the young buyer's use. This clause makes an already difficult to enforce law even weaker. In Britain some salespeople even open packets of cigarettes and sell them singly to children. If a law preventing sales to minors is made, it is important that it should be enforced – which can be extremely difficult.

Belgium, Finland, France and Canada amongst other countries have legislation to reduce the chance of children using cigarette machines. For example, the machine is under supervision at all times. In Belgium, Bulgaria, Finland, France, the Federal Republic of Germany, Italy, Poland, some states of America, Ivory Coast, Libya, Iraq, Jamaica, Japan, U.K., and several other European countries have actually made laws regarding under-age smoking. These legislations usually apply to smoking in and around school premises, but in the U.K. there is actually a law – rarely enforced – whereby the police are required to seize tobacco or cigarette papers from children under 16 years of age if they are smoking in the street or public places.

Another aspect of legislation, not directly related to children, but clearly affecting them is increased taxation on tobacco products putting them out of reach of children's finances.

Advertising

Unfortunately in many countries of the western world, and a rapidly increasing one in countries such as India, is advertising of the tobacco industry. As tobacco sales fall in the west, the tobacco industry is attempting to boost its sales in the east where cigarette smoking was formerly the exception rather than the rule (25). A look at Indian magazines, such as *Jet Set* for young people, or *Flair* for women, reveals cigarette advertisements showing happy young people in sports or holiday surroundings. Restrictions in many countries now forbid this

direct appeal to young people and women and the glamorous or tough image cannot now be used.

There is no doubt that children – and adults too – take in a lot from advertising. Chapman (1) has given a very valuable analysis of the effects of cigarette advertisements clearly showing how the illustrations in cigarette advertisements are decoded to give important messages to the observer as to what the cigarettes will do (26), for example, offering friendship and social confidence and independence. Children who could name a favourite advertisement and therefore had looked carefully at it were significantly more likely than other children to have positive views about the value of smoking (27). Children, especially smokers, and probably those who are considering starting to smoke, believe that smoking calms one's nerves, gives one confidence and keeps one's weight down. These are all messages which are contained in current cigarette advertisements in Britain. The image of toughness, now banned, had largely vanished from children's concept of smoking, but has been replaced by the aforementioned psychological or physiological messages.

Over a dozen countries have banned all cigarette advertising and over thirty countries have banned cigarette advertising on radio and television. Several countries prohibit advertising directed at young people.

Advertising of cigarettes must be controlled in India. There are more than enough tobacco problems to overcome without this additional menace.

Need no. 10 : Legislation is needed on many aspects of tobacco production and sales in order to influence the macroclimate of tobacco acceptance.

Conclusions

There is a need to undertake surveys, utilising a standardised simple completely anonymous questionnaire administered regularly to children and teachers to assess tobacco usage of all kinds at all ages.

Below are enumerated the possible influences on the micro- and macroclimate of children's tobacco usage. These need to be influenced in order to create "Tobacco-free generation by the year 2000"

Possible influences on the micro-climate

1. Evidence of the effects of parental tobacco usage on children.
2. Involvement of parents and the whole community in all tobacco intervention programmes with children.
3. Help for teachers to stop using tobacco at least in front of the children.
4. Development of a school policy (perhaps national) with regard to pupils' and teachers' tobacco usage on school premises. This policy must be known to all.
5. To have available to all teachers, free or cheap guidance and materials for

teaching.

(a) an all round approach including health risks; social aspects; resisting social pressures;

(b) materials for parents in order to make a whole family approach to the problem. (U.I.C.C. etc.)

6. Help for pupils who want to give up the tobacco habit.

Possible influences on the macro-climate

1. Government action to plan new crops to replace tobacco.
2. Legislation as recommended by the U.I.C.C.

(a) There should be no advertising or sales promotion of tobacco products (including sports sponsorship by the tobacco industry).

(b) There should be no smoking (or other tobacco usage) in any part of any educational establishment where young people are likely to be present.

(c) Young people should as far as possible be prohibited from smoking or using tobacco in public places, with especial emphasis on educational establishments.

(d) It should be illegal to sell or offer tobacco products to children or adolescents. The crime should be that of the vendor not the buyer.

(e) Cigarettes (and other tobacco products) should not be sold from vending machines where there is a possibility of unsupervised access by young people.

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Study of Tobacco Habits in School Children in Goa

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Children record everything that happens around, very often without knowing the significance, but which has profound influence on their behaviour later, when they grow up. There is hardly a child who does not know the smoking and chewing habits of their parents and teachers and there is hardly any parent or a teacher who knows the tobacco or for that matter probably any other habits of their children or wards. A survey conducted in nine schools in villages of Goa, India, covering 1668 children by self-administered structured questionnaire revealed that 18% were tobacco habitues. The prevalence of tobacco habit was higher in boys (22%) than in girls (13%). The common habits were the use of "mishri" or "masheri" (i.e. roasted and powdered tobacco rubbed over the gums with the index finger) and "creamy snuff" toothpaste. While 84% (256) of the habitues had a single habit, 12% and 4% had double and triple habits respectively. The mean age of acquiring the habit was 11.9 years. Almost 75% of the habitues stated family influence as the commonest influencing factor.

Introduction

In a survey of tobacco habits, in Goa, in persons 15 years of age and above, it was observed that 40% of those with tobacco habits had acquired it by the age of 20 years. This percentage rose to about 60% by the age of 25 years. The common tobacco habits were chewing tobacco, smoking cigarettes or bidis, using "masheri" also known as "mishri" and tobacco "creamy snuff" toothpaste. Masheri is powder of dried and roasted tobacco leaves, sometimes mixed with ash and rubbed over the gums with index finger. Creamy snuff is a toothpaste whose main constituent is tobacco and is sold under various brand names like "Dentobac", "Ipco", "Tona" and "Amino".

Materials and methods

It was therefore decided to study the prevalence of tobacco habits of all four types in school children. High schools were approached for their co-operation. A proforma with nine questions which could be easily answered by children of ages 10 years and above, was distributed in the class. The students were assured of confidentiality of information and they were not required to enter their names on the proforma. The filled up proformas were collected back in the classrooms.

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All English medium high schools from randomly selected villages of Goa were entered in the study. The average number of children in a class was forty. 1668 students filled up the forms. The students had not been exposed to any anti-tobacco programmes prior to the survey. In one school where the teacher of the class had discussed it earlier, the percentage of positive answers were minimal and hence that class was not included in the analysis.

Habitue was defined as one using any of the above mentioned tobacco products continuously for atleast six months, with a frequency of not less than once a day.

Results

Table 1 gives the distribution of male and female students and the habitues among them by age. The study group consisted of 56% boys and 44% girls. Majority (90%) of students were between the ages of 10 and 17 years, and 10% were above 17 years.

Table 1. Age and sex-wise distribution of all students and those with tobacco habit

Age in	Male students			Female students			Total students		
		with habit			with habit			with habit	
years	No.	No.	(%)	No.	No.	(%)	No.	No.	(%)
10	9	1	(11)	9	3	(33)	18	4	(22)
11	29	6	(20)	24	1	(5)	53	7	(13)
12	77	10	(13)	72	5	(7)	149	17	(11)
13	151	31	(20)	107	12	(11)	258	43	(17)
14	158	40	(25)	132	20	(15)	290	60	(21)
15	160	36	(22)	169	21	(12)	329	57	(17)
16	155	29	(18)	112	21	(19)	267	50	(19)
17	84	23	(27)	53	10	(19)	139	33	(24)
18	66	19	(28)	40	3	(8)	106	22	(21)
19	36	12	(33)	8	0	–	44	12	(27)
20	10	2	(20)	2	0	–	12	2	(17)
21	5	0	–	0	0	–	5	0	–
Total	940	209	(22)	728	96	(13)	1668	305	(18)

Out of 1668 students 305 (18%) had tobacco habits. Of the 940 boys 209 were habitues i.e. a prevalence rate of 22% and of 728 girls 96 were habitues giving a

prevalence rate of 13%. There were few students at ages 10, 11 and 19 to 21 to arrive at a reliable estimate of prevalence of habitues at these ages. It can be seen from Table 1 that the percentage of tobacco users increased gradually with age from a minimum of 11% at the age of 12 years to a maximum of 27% at the age of 19 years.

Table 2. Distribution of tobacco users by type of habit

Habits	Males (n = 209)	Females (n = 96)	Total (n = 305)
Masheri	102 (49)	62 (65)	164 (53)
Dentobac *	107 (51)	52 (54)	159 (52)
Chewing	32 (15)	12 (13)	44 (14)
Single	183 (87)	73 (76)	256 (84)
Double	20 (10)	18 (19)	38 (12)
Triple	6 (3)	5 (5)	11 (4)

Figures in parenthesis are percentages and do not add to 100 in the upper half of the table because of multiple habits.

* Brand name of "creamy snuff" toothpaste, which has tobacco as the main constituent.

Among the habitues, as seen in Table 2, masheri use was found in 53% and proportionately more females than males had this habit. The use of creamy snuff was the next frequent habit and was present in 52% of habitues followed by chewing habit in only 14% of habitues. While 84% of habitues had a single habit, 12% and 4% had two and three habits respectively. Smoking habit was not common and was indulged in "for fun" by the students. Thirty-eight students (27 males and 11 females) stated that they had smoked for fun. Only one male student out of 940 was a regular smoker.

From Table 3 it is seen that only 161 out of 305 students with habit answered the query on the influencing factor. Almost 25% of the students, both boys and girls were influenced by their fathers. About the same proportion of girl students were influenced by their mothers and brothers while for boys the next important influence after father was that of bother and friend. A very small proportion (4%) of students were influenced by their teachers.

Only 221 (72%) of habitues stated the age at which they started the habit (Table 4). The earliest age of start was given by a boy as 3 years and 7% of students

Table 3. Influencing factor in tobacco users

	Males	Females	Total	(%)
Father	27	15	42	(26)
Mother	13	13	26	(16)
Brother	18	14	32	(20)
Sister	1	10	11	(7)
Uncle	6	4	10	(6)
Friends	17	2	19	(12)
Teachers	5	2	7	(4)
Others	11	3	14	(9)
Total	98	63	161	(100)

Table 4. Age at start of tobacco habit

Age in years	Number	(%)
5 and below	16	(7)
6 and 7	20	(9)
8 and 9	15	(7)
10 and 11	51	(23)
12 and 13	51	(23)
14 and 15	49	(22)
16 and 17	17	(8)
18 and 19	1	(1)
20 and 21	1	(1)
Total	221	(100)

had started their habit by the age of five. However a large majority (68%) of students started the habit in their early teens, so that by the age of 15, 91% of habitues had acquired the tobacco habit. Mean age at start of habit was 11.9 years.

Table 5 gives the duration of different habits. On an average 72% of habitues gave this information. It is seen that a large proportion (44%–75%) have had the habit for a duration of 3 years or less. However, as much as 25% of masheri users have had the habit for durations varying from 8 to 16 years. Whereas only 6% of creamy snuff users and 11% of chewers have had the habits for long durations (8 to 11 years).

Table 5. Duration of habits

Duration in years	Masheri (n = 111)	Creamy snuff[+] (n = 126)	Chewing (n = 28)
	%	%	%
0– 3	44	56	75
4– 7	31	39	14
8–11	18	6	11
12–16	7	–	–

[+] Toothpaste with tobacco as the main constituent.

Discussion

Few studies on prevalence of tobacco habits in children have been undertaken in India. Most of the studies from the Western countries are with respect to the

habit of smoking in children. These studies have reported that mean age at which children start to smoke vary from 11 to 15 years (1). The use of tobacco in forms other than smoking is more prevalent in South-Asian countries.

While all the students participating in the present study responded to the questionnaire, the response of habitues of different types varied. While 79% of creamy snuff users (126/159) declared their age at start, only 67% (111/164) of masheri users and 63% (28/44) of tobacco chewers declared their age at start. While 65% (63/96) of female habitues answered about the influencing factors only 46% (98/209) of male habitues answered the same question. A more thorough study is required to fathom the cause of differential response. However, it may be inferred that tobacco paste is a more well defined product-well packaged, available as an entity in the market and has to be purchased as a distinct item as against chewing tobacco or masheri-which is not a packaged, well defined item and is constantly available at home because of use by the elders.

The prevalence of type of habits was not markedly different between boys and girls. Prevalence of both masheri and tobacco paste was a little higher in boys (11%) than girls, (9% and 7% respectively). The prevalence of chewing habit was almost the same, 3% in boys and 2% in girls. The double or triple habits, however, were more frequent in girl habitues (24%) than boy habitues (13%).

For the boys with habit the most important influencing factors were father, brother and friend while only one stated to be influenced by sister. Brothers had almost equal influence on boys as well as girls whereas the sisters had very little influence on boys with respect to the tobacco habit behaviour. The peer pressure outside the family appears to wield greater influence with boys than girls. Overall, from the 52% of habitues that responded to this question it is seen that almost three quarters take to the habit because of family influence. The mean age at start was 11.9 years. Sex-wise analysis of age at start and duration of habit was not undertaken as the numbers were not large enough.

The study indicates the different patterns of habit in boys and girls and the different influencing factors. It is obvious that indepth study and more extensive coverage is required to study the various factors which influence the young minds to take up the habit, though one may conclude that family environment is foremost in moulding or introducing a behavioural pattern in the rural children. This has important implications in the methodology of educational programme. Unless the family environment and adult education is undertaken along with school education, the effect on the pattern of tobacco usage may not be significantly altered.

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Section IV

Action on Tobacco Control

Tobacco and Health : The Indian Scene. L.D. Sanghvi and Perin Notani (Eds.)
UICC Workshop, Tata Memorial Centre, Bombay 400012, India. (1989)

Smoking Control Policy

David Simpson[1]

Introduction

The World Health Organisation, the International Union Against Cancer, the Royal College of Physicians of London and many other expert organisations which have examined the problems of tobacco and ill health, have made numerous recommendations about how tobacco should be controlled in order to reduce the disease, disability and premature death which it causes. Among the measures which are consistently recommended are :

1. Stopping the promotion of tobacco products;
2. Public education and public information programmes;
3. Raising price through taxation;
4. Health warnings;
5. Reducing the emission levels of toxic components;
6. Controlling smoking in public places.

This paper examines the main components of a tobacco control programme, with particular reference to those which are likely to meet resistance from tobacco interests.

The smoking history of the United Kingdom provides useful background experience for the examination of smoking control policy. Unfortunately, the UK was among the first countries to adopt widespread cigarette smoking and could be said to have led the world in this most disastrous cause of ill health. Indeed, as a direct result of this, the UK still has the highest lung cancer mortality rates in the world.

Starting first as a habit among males, cigarette smoking rapidly gained acceptance and by the 1950's, the majority of males and around half of all females were regular cigarette smokers. The upward trends continued until 1962, when the Royal College of Physicians of London published its first report (Fig. 1). This summarised scientific evidence which demonstrated beyond doubt–and publicised to the nation as a whole–the fact that smoking was a major cause of disease. Male smoking habits soon began to decrease and although women were slower to follow this trend, smoking in both sexes is now steadily declining, probably faster than in any country in the world.

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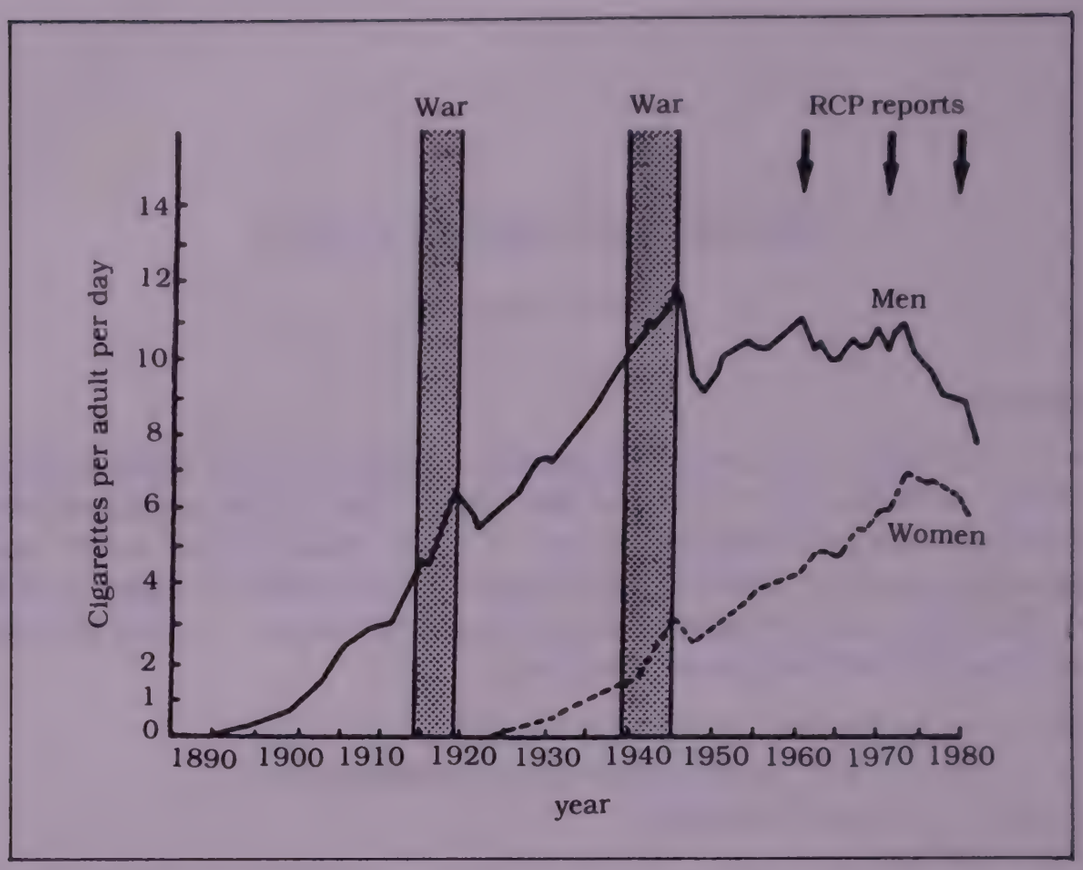

Fig. 1. Tobacco consumption in the UK 1890 to 1981, given as average number of cigarettes per adult per day for men and women separately, irrespective of whether they smoke or not. The arrows indicate the dates of the three previous Royal College of Physicians reports. Data from Tobacco Research (now Advisory) Council [reference 1 and unpublished data reproduced with permission].

Two important examples of smoking control factors are demonstrated in the UK's smoking history as shown in Fig. 1. Firstly, the temporary drop in consumption after each of the two World Wars illustrates the effect of economic factors : in the first instance an economic slump and in the second, a similar adjustment of the post-war economy coupled with a large rise in tobacco tax in 1947, had a major impact on consumption.

Secondly, the delivery to the public of detailed and unequivocal information about the dangers of smoking in the first report by the Royal College of Physicians in 1962 ended the steady rise of per capita consumption and set the course for the decline of smoking in Britain. These two aspects of smoking control will be dealt with in further detail in the sections dealing with raising price through taxation and public information programmes.

Banning tobacco promotion

In India, the topic of tobacco promotion is mainly concerned with cigarettes, rather than bidis, oral tobacco or any of the other Indian tobacco products.

All cigarettes are intrinsically the same : tubes of paper with chopped dried tobacco leaves inside which when ignited, produce smoke containing several thousand chemicals. Many of these are known carcinogens and other highly toxic compounds. It is virtually entirely due to advertising that one brand of cigarettes is perceived as having different characteristics from another.

Cigarette advertising never draws attention to the toxic chemicals or the unique and substantial risks involved in inhaling the smoke from a cigarette. Instead, advertisements are exclusively concerned with creating an image intended to appeal to a particular group of people. We know from information provided from within the advertising industry, some of it inadvertently or reluctantly, that strenuous efforts are made to aim certain advertisements at young people; to reassure smokers about their fears of the health risks of smoking; and to associate smoking in general and individual brands in particular, with positive concepts and images.

Above all, tobacco promotion maintains the social acceptability of tobacco use. Health education cannot deliver its message to the public – especially to young people – at face value so long as huge sums are spent each year associating cigarettes with success, glamour, independence, sports and all sorts of other desirable things.

Now that cigarette consumption is declining in many industrialised, Western countries, the tobacco companies are increasingly turning to the developing world to increase their sales there. Advertising and other promotional techniques used in developing countries are often outrageous in content and breach all standards which the companies claim to observe in those Western countries which have taken action to control promotion.

Another important feature of advertising in developing countries is its aim of increasing the number of consumers. The industry will always argue that advertisements are only designed to make customers switch from one brand to another. But if advertising was not used to increase smoking as well as promote brand-switching, it is difficult to see why it would take place in countries with tobacco monopolies, like Kenya and Mauritius. In Kenya, the holder of the monopoly, or near-monopoly of the cigarette market, a subsidiary of BAT, was the fourth largest advertiser for any product in the late seventies.

An essential feature of a successful policy to prohibit the promotion of tobacco is that the regulations should be backed by legislation. The tobacco industry will always try to persuade governments that measures implemented by voluntary agreement between the industry and the government will be better. Experience shows that they will indeed be better for the industry, but will be far from effective. The experience of the UK is a sad testimony to this fact, where successive voluntary agreements on advertising have merely led to more examples of tobacco

industry ingenuity in circumventing the intentions of the government.

The shortcomings of voluntary agreements on tobacco advertising are as follows :

1. They tend to be highly selective in nature : many significant types of promotion are excluded from being regulated.
2. Different government departments are involved, often pulling in opposite directions. For example, the ministry responsible for sports would like as much sponsorship of sport from private industry as possible, but tobacco sports sponsorship will have precisely the opposite effect to the efforts of the health ministry to try to ensure that tobacco is not linked with healthy and exciting qualities associated with sport.
3. The voluntary nature of the system means the companies have little to lose by breaching the agreement and nothing at all to lose by refusing to agree to more stringent measures, so long as no real threat of legislation exists.
4. The many rounds of meetings involved with ministers, officials and other organisations (advertising industry, retailers, etc.) mean that negotiations for new voluntary agreements can take years, thus giving plenty of time to plan how to circumvent the restrictions being negotiated.
5. Being one of the parties to the voluntary agreement on cigarette advertising, the health ministry is effectively neutralised. From being the obvious point from which improvements, from the health point of view, could be initiated, encouraged and executed, the health ministry is forced into the position of arbiter between health on the one side and the tobacco industry on the other.

Even where bans have already been implemented, the industry still tries to get round them. In the Sudan, for example, a ban on all tobacco promotion in 1984 resulted in advertisements for Marlboro cigarettes being very slightly changed to become advertisements for Marlboro cigarette lighters. The changes left virtually the entire image intact, including the distinctive red chevron, Marlboro name and typeface, together with the "macho" cowboy and his horse. After a threat from the government to invoke the law, these advertisements were removed.

In Norway, where all forms of promotion have been banned since 1975, Camel cigarettes attempted to get round the law by advertising a new product : Camel boots. The first advertisements in a series of attempts employed the same actor familiar worldwide for advertising Camel cigarettes, in a typical rugged, outdoor setting. Characteristically paddling his canoe, he was ostensibly advertising a boot, one of which could just be seen.

The Norwegian Government's National Council on Smoking and Health threatened the company with legal action, whereupon the advertisement was modified. This time, more of the tough man's boots were visible, together with a superimposed boot shown in the foreground, but the distinctive Camel name and logo were still above the picture. Once again, legal action was threatened. A third advertisement was produced, using the same actor but in a different setting – this

time clad in his Camel boots, he appeared to be trying to haul a heavy load up a steep ramp. A somewhat different typeface had been used for the name "Camel" and it was set straight rather than in a curve, but in the usual colour. Undeterred, the government again threatened legal action and at this stage the company gave up.

In both the Sudan and Norway all tobacco promotion is banned by law. In each case, a multi-national tobacco company tried to circumvent the law but backed down when legal action was threatened. In short, legislation works.

Raising price through taxation

It is widely accepted in the United Kingdom that regular increases in cigarette taxation have been the most effective single measure causing the steady decline in consumption in recent years.

In virtually every country, no matter what type of economy it has, the government can go on increasing taxation in real terms with the combined result that consumption will fall, but the government will still make more money, in real terms, from taxation. This point is well illustrated by the experience of the United Kingdom in recent years as is demonstrated by the two graphs. Figure 2 shows that consumer expenditure on cigarettes, adjusted for inflation, varies according to the price of an average packet of twenty cigarettes. Most of the price increases in the period shown were caused by tax increases.

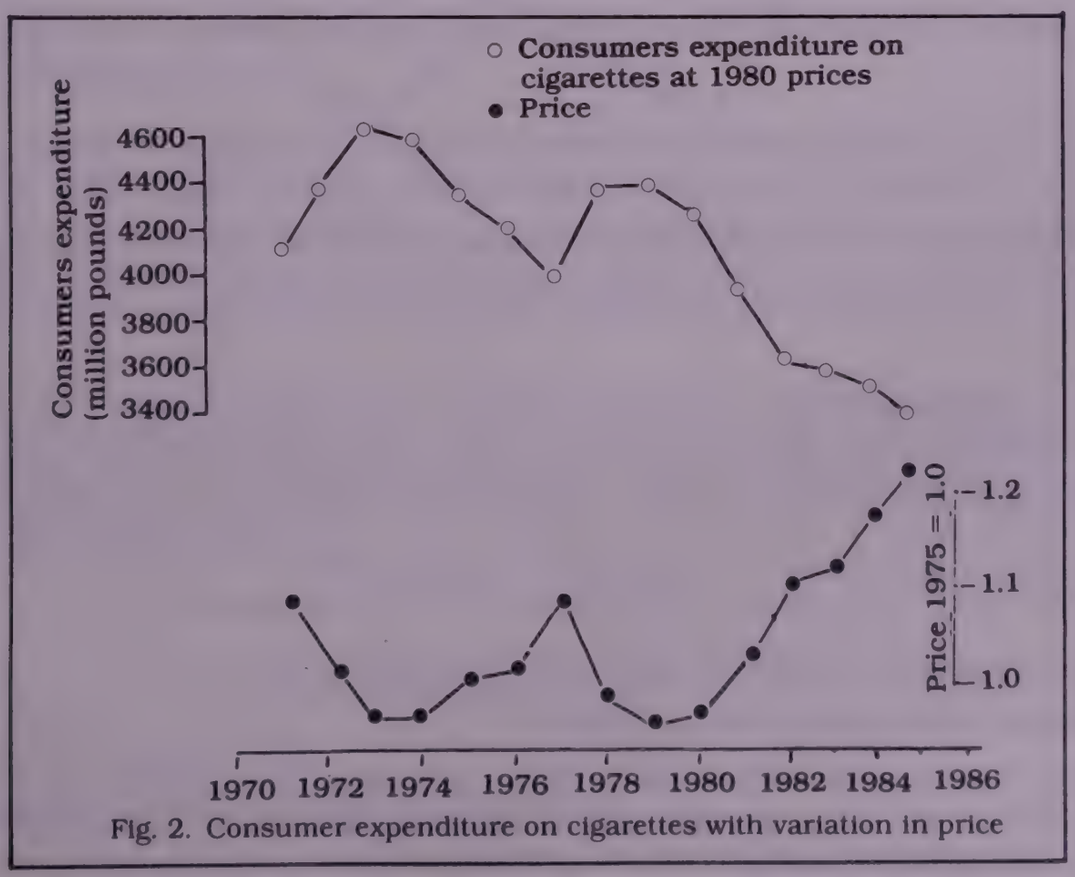

Fig. 2. Consumer expenditure on cigarettes with variation in price

Figure 3 shows the price curve together with the government's income from tobacco taxation, also adjusted for inflation. It shows clearly that the government's income increases as the price rises; but when the price is allowed to fall, even though consumers spend more money on cigarettes, the government's income falls too. Eventually a point of diminishing returns would be reached, but even in the United Kingdom where prices are comparatively high and consumption has been falling for some years, economists say that this point lies many years in the future.

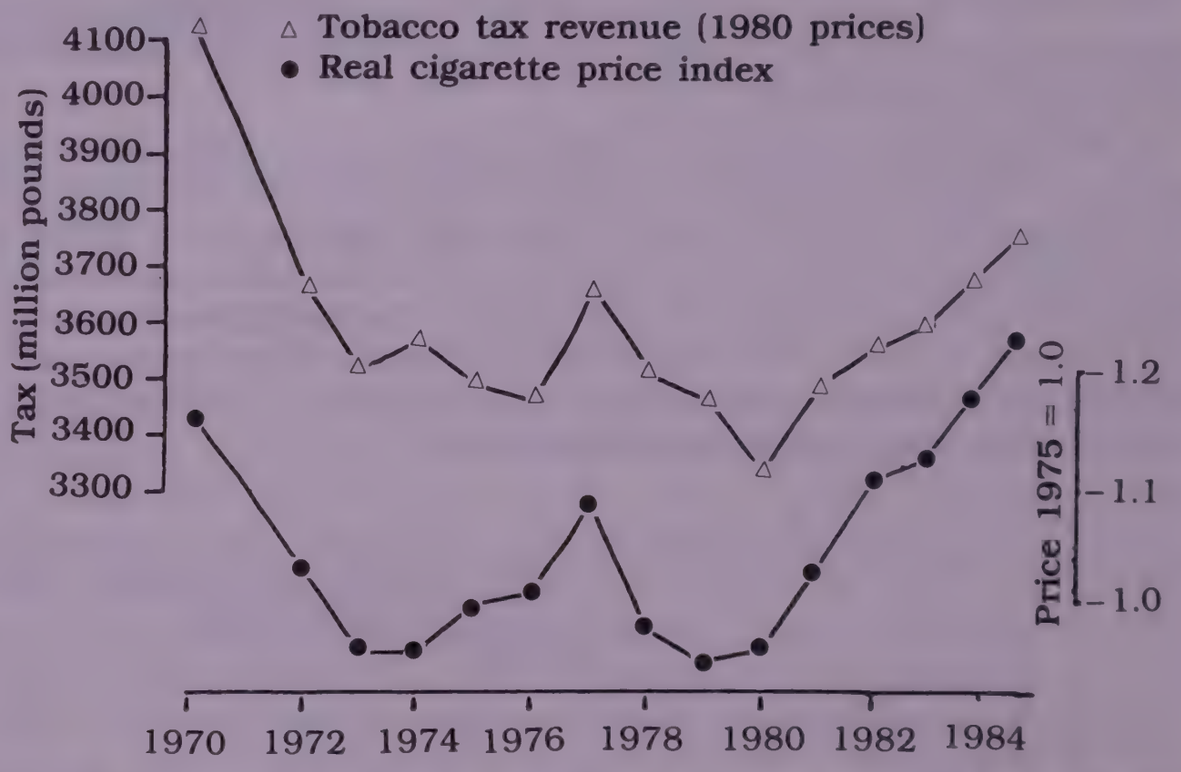

Fig. 3. Government's income from tobacco taxation adjusted for inflation

Economists have also demonstrated that an increase in tobacco tax is borne more by the wealthier smokers than the poorer ones. This is because the smoker who can more easily afford the new, increased prices is more likely to maintain the same smoking habits after a rise in price; but smokers with less money will be more likely to give up or cut down their consumption.

Public education and public information programmes

Firstly, some definitions are offered :

Public education work consists mainly of education programmes which are aimed at specific target groups, such as school children of various ages; young adults; and adult smokers.

Public information programmes are aimed at providing a continuous, general flow of accurate and interesting smoking control information, directed towards people via the news media and other existing channels of information.

Public education and public information programmes often overlap; and both are essential components of a comprehensive smoking control programme. They do not compete with each other, but rather complement each other and reinforce each other. Each will now be examined in more detail.

Public education programmes

Among the main objectives of public education campaigns are :

- To maintain the behaviour of nonsmokers and to change the behaviour of smokers;
- To change the cultural background of society against which cigarette smoking may be viewed as a normal activity; and to establish the realistic view that cigarettes are dangerous to health;
- To establish nonsmoking as the normal behaviour and to ensure the right of the nonsmoker to smoke-free air.

Looking first at programmes aimed at children and adolescents, such programmes are composed of two parts :

- A national programme, involving all segments of society to arrange, as far as possible, that children grow up in a nonsmoking atmosphere;
- Specific school or community programmes, to reach special target groups and to attack specific problems.

The first step in any such programmes will usually be to gather baseline data of current smoking practices and attitudes. Leaders of programmes must be chosen for their special knowledge of children and their ability to communicate with them. Training sessions for leaders may be necessary, to familiarise them with the problems of smoking and the materials prepared for the smoking education programme.

Maximum effectiveness of any smoking education programme will be obtained with a plan for reinforcement once the programme has been completed, and evaluation of programmes will be essential to assess their effectiveness and to plan for future programmes.

The design of a smoking education programme may be considered as a series of steps :

- Analysing the problems to be tackled;
- Identifying target groups;
- Designing materials suitable for each group;
- Pre-testing the material on small groups and correcting any faults;

- Co-ordinating all the groups – such as teachers – who will deliver the programme; and ensuring that suitable materials are available to them;
- Delivering the programme to the target group – the teachers actually conducting sessions with the pupils;
- Evaluating the effectiveness of the programme.

Public information programmes

As noted earlier, there is obviously much overlap between the process of conveying information to specific target groups through public education programmes and the regular transmission of information to the public as a whole through public information work. Nevertheless, a public information programme is important for helping to reinforce the more specific education programmes.

The most important characteristic of a public information programme is the fact that it makes use of radio and television, newspapers, magazines and other news media, to convey information to very large numbers of people. The success of such a programme therefore depends on a good understanding of the way the press and broadcasting organisations operate; and it will be helpful to maintain good relationships with journalists, to be readily available to supply information and comment and to have suitable experts available to assist journalists whenever necessary.

The objectives of a public information campaign include the following :

- Increase public awareness of the health consequences of smoking;
- Persuade adults to give up smoking by rational argument;
- Influence youth;
- Create an atmosphere in which it is realised that smoking is an unhealthy behaviour;
- Establish the rights of nonsmokers to breathe smoke-free air;
- Publicise specific policy objectives in order to mobilise sympathetic public support for them – for example with regard to tobacco taxation;
- Analyse and criticise aspects of tobacco industry activity;
- Counter inaccurate information with true facts and figures about smoking;
- Discuss,emphasize and reinforce the importance of other anti-smoking programmes.

Various elements of public information work are of particular interest to press and broadcasting media. For example :

- The medical and scientific case against smoking;
- Changes in trends in smoking consumption;
- Smoking amongst exemplar groups, such as doctors, teachers, sports athletes and so on;

- Ways of giving up smoking;
- Government action against smoking;
- What schools are doing about smoking;
- The rights of nonsmokers;
- The special problems of women and smoking;
- Special nonsmoking days or weeks – such as National No Smoking Day in the UK.

Health authorities in the UK are in no doubt that public education and public information programmes have been a major cause of this decline. It must be emphasized that other factors, especially taxation increases in recent years, have been very important in bringing about the decline; but there is no doubt that each type of activity supports the other.

Health warnings

All packages for tobacco products and, where advertising is still permitted, all advertisements, should carry strong warnings about the effects of tobacco on health. Such warnings are obviously of limited value where a significant proportion of the population at risk is unable to read the warnings. In countries where a number of different languages are used, the tobacco industry will always try to use the language understood by the smallest number of people. Furthermore, it will use every effort to make the warnings ineffective, by trying to persuade the government not to use legislation to impose the warnings, by trying to dispute the proposed wording of the warnings, substituting less effective messages and by reducing the impact of the messages by the addition of lengthy and distracting attributions and the use of poor quality typefaces, etc.

Legislation is thus essential for effective health warnings. Otherwise, the industry will never agree to any warnings which will have a significant effect on sales; and once a mediocre system has been adopted, the government will find it exceedingly difficult to negotiate improvements.

In addition, ideally there should be a system of rotating warnings whereby several different messages are in use at any one moment, appearing at random on all packages and, if they are still permitted, on advertisements. Sweden and Norway each use sixteen warnings; and periodically, all sixteen are replaced with a new set, to ensure that the messages do not become ineffective by being too familiar to consumers.

As with all individual aspects of a tobacco control policy, health warnings alone will not solve the tobacco problem. But they can be an important complement to public education and information work; and they provide daily evidence to tobacco users of the seriousness which their government attaches to the problem.

Messages should mention specific diseases caused by tobacco use; they should attempt to convey the magnitude of the risk compared to other risks which

people take; and the unique nature of the risks of using tobacco, which is dangerous at virtually any level of use, rather than simply being dangerous when abused, as is the case with many other dangerous substances or habits. The messages should be expressed in language which ordinary people can understand. In short, health warnings should be devised along exactly the same lines which a manufacturer uses when devising positive marketing messages to appear on packages or advertisements.

Reducing the emission levels of toxic components

This is a complex area of policy and it is not proposed to cover it in detail here. The vast majority of experience and all the recommendations of international expert groups relate to tobacco smoking and in particular to manufactured cigarettes. More work needs to be carried out on the toxicity of other types of tobacco use, of which there is probably no greater diversity than in India, and the opportunities for lowering the risks to Indian tobacco consumers. It is encouraging to note the pioneering work in this field which has already been carried out in Bombay.

Suffice it to say that lowering the tar emission levels of cigarettes has been shown to significantly reduce the incidence of lung cancer caused by smoking; and a number of Western governments are also attempting to reduce carbon monoxide yields in view of the evidence which suggests that carbon monoxide plays a role in the etiology of heart disease in smokers. Nicotine emission levels have also been reduced in the West, though below a certain nicotine level habitual cigarette smokers may smoke more cigarettes to compensate. A low tar, low carbon monoxide, but medium nicotine cigarette has therefore been suggested as the least dangerous product for those who smoke.

Nevertheless, it should be stressed that this area of tobacco policy should never be seen as anything but an interim measure to be taken while every effort is made to eliminate all use of tobacco. While lower tar cigarettes may lower the risk of lung cancer, this lower risk still remains far larger than any risk which could be considered acceptable; and there is no conclusive evidence that tar reduction lowers the substantial risks of cardiovascular disease and chronic obstructive airways disease caused by smoking.

On no account should the tobacco industry be permitted to determine the way emission policy is implemented. Furthermore, it should be borne in mind that if the public is educated about the relative dangers of low tar and high tar cigarettes, there is always a danger that continuing smokers will persuade themselves that so long as they smoke a low tar brand, they will be "safe".

In countries like India, significant reductions of future lung cancer mortality could be effected by setting upper limits for tar levels and regularly reviewing and lowering them. Sales-weighted tar levels can be reduced relatively quickly and cheaply, especially if the government takes legal powers to enforce compliance to the limits. Differential tax rates favouring the lower-tar brands may be considered, a system which was used successfully in the UK.

Controlling smoking in public places

This is the area which the tobacco industry now fears more than any other in the Western countries, in terms of being an effective influence in reducing cigarette consumption. In developing countries its potential may at present be less than the other main smoking control measures, but nevertheless every effort should be made to encourage the provision of smoke-free areas in all public places. In particular, employees should be able to work free from other people's smoke. Ultimately, nonsmoking should be the norm, with smoking areas provided where convenient, rather than the other way round, as at present.

Further reading

For a much more detailed consideration of smoking control policy than can be given here, a number of reports by international agencies are available. In particular, the following are recommended :

Guidelines for Smoking Control (2nd edition). International Union Against Cancer, Geneva. UICC Technical Report Series – volume 52.

A Manual on Smoking and Children. International Union Against Cancer, Geneva. UICC Technical Report Series – volume 73.

Controlling the smoking epidemic. Report of the WHO Expert Committee on Smoking Control. Technical Report Series 636.

Smoking control strategies in developing countries. Report of a WHO Expert Committee. Technical Report Series 695.

Tobacco and Health : The Indian Scene. L.D. Sanghvi and Perin Notani (Eds.)
UICC Workshop, Tata Memorial Centre, Bombay 400012, India. (1989)

Action on Control of Tobacco : Role of State Government

S.M. Bhadkamkar[1]

Introduction

It is now a well established fact that consumption of tobacco is responsible for three major health problems viz., cancer, heart diseases and respiratory diseases. Therefore, control of tobacco will help to reduce the mortality and morbidity due to these conditions to a considerable extent. It is accepted that tobacco is the most important single causative agent which is totally preventable and banning its use will result in alleviating innumerable human sufferings.

As far as cancer is concerned, tobacco is responsible for cancers of oral cavity, upper alimentary tract and respiratory tract. Therefore, control of tobacco should become one of the most important preventive measures to be vigorously pursued by Public Health Department of every State.

Actions to control tobacco will consist of :

i) Control over the production of tobacco,
ii) Control over the consumption of tobacco.

Control over the production of tobacco

It is said that India ranks third in the world as regards tobacco production and 280 million kgs. of tobacco is consumed per year in our country. The subject of controlling production is linked up with financial implications and agricultural production policies which mainly come under the perview of the Central Government.

Control over consumption

Modes of consumption

Tobacco is consumed both by urban and rural population of the State, in following ways :

1. *Smoking* : It is estimated that 80-86% of tobacco is consumed for smoking, either as bidi or as cigarettes.

   a) *Bidi* : In our State, bidi is more popular especially among lower socio-

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economic class and rural population. This is more important because carbon monoxide level in bidi smoke is much higher than that in cigarette smoke as tendu leaf used as a wrapper for bidi is much less porous than cigarette paper.

b) *Cigarette* : Most of the people smoke cigarettes without filter. It is common among higher socio-economic class mostly from urban areas.

2. *Chewing* : Almost 13% chew tobacco either with pan or lime. This is common both among urban and rural population.

3. *Mixed consumption* : There is a small section of 15% persons who are addicted to both the habits of chewing and smoking. This group though limited, is epidemiologically important because they have higher relative risks for development of cancer, than those who either only chew or smoke tobacco.

4. *As powder* : Only 1% to 3% use tobacco either in the form of snuff or dental paste powder.

In Maharashtra, there are a number of bidi factories mainly concentrated in Nasik and Ahmednagar districts. Tobacco is grown in border areas of Kolhapur like Nipani.

Action to reduce consumption

Though the State Health authorities were aware of the health hazards of tobacco consumption, no systematic drive or programme was undertaken till recent past. During 1986-87, however, the State had taken steps in the right direction as follows :

1. *Launching of state oral health and cancer control programme* : As stated above, prevalence of oral cancer and cancers of respiratory and alimentary tract is high in tobacco users. Oral cancer is one of the three important problems to be tackled under the Oral Health Programme. Similarly, cancers of other organs attributable to tobacco usage are an important part of the Cancer Control Programme. A National Cancer Control Programme is already accepted and National Oral Health Programme is said to be under consideration. However, both these programmes are not fully operative in any State in the country. The State of Maharashtra has taken lead in this direction and have appointed Working Groups to evolve a State policy for Oral Health as well as Cancer Control Programme. It is expected that not only the policies will be finalised within the next few months, but an action programme for implementing them will be fully operative. In fact, the State has already started taking some action in this regard, in anticipation.

2. *Making existing legal provisions operative* : Under Section 116 of the Bombay Police Act 1951, smoking and spitting is prohibited. This provision was not made fully operative till now. This provision can be applied to all public and government institutions, buildings and offices. The powers to implement it is with the authority in-charge of the institution. It has been

decided to fully implement this provision in the State.

A beginning is being made with implementation of this provision in health organisations and following instructions have been issued :

a) To ban smoking in the hospital buildings.
b) To ban smoking in the hospital premises.
c) To encourage health staff to give up smoking.
d) To ban sale of tobacco in the canteens attached to hospitals.
e) To display posters and slogans in the hospital premises against smoking.
f) Same instructions to be applicable to all the offices of the Health Department. No ash trays to be maintained.
g) Health staff to refrain from smoking while on tour or in parties. In short, by own example, health staff should give momentum to anti-tobacco campaign.

3. *Creation of public awareness :* This year, the State Government has earmarked an amount of Rs. 5 lakhs for Health Education in Cancer which will mainly include anti-tobacco activities as follows :

a) Posters and stickers mainly against smoking have been prepared and distributed.
b) Catchy slogans, e.g. "Your smoking is injurious to your health", etc. are selected for wall painting on a large scale.
c) State Transport Department has been approached to make non-smoking zones effective and also to ensure their implementation.
d) State Government has also approached Government of India undertakings like Central and Western Railways and Indian Airlines to reduce smoking zones permitted and enlarge non-smoking zones.
e) It is decided to utilise mass media like television, radio, newspapers for creating awareness.
f) Action to help smokers :
   - Persuade smokers to give up smoking by utilising services of those who were chain smokers but have given up smoking.
   - Till the smoker gives up smoking, he should be persuaded to at least reduce number of puffs per minute so that harm done will be minimum. It has been observed that Indian smokers take at least 2 puffs per minute compared to 1 puff in the west.
   - Till the smoker gives up smoking, he will be persuaded to use cigarettes which are less harmful, i.e. with good filteration.
   - If a person has got mixed habits of smoking and tobacco chewing, he should be persuaded to give up one initially to be followed by the other.
   - During routine programmes persons who have pre- cancerous lesions

like white patches in the mouth will be warned about cancer and persuaded to give up use of tobacco, an advice which is likely to be heeded in view of threatened danger.

Implementation of anti-tobacco strategy

Organisation

As already stated above, the State is already taking steps to launch full fledged State Cancer Control and Oral Health Programmes. In order to formulate the policies and review the progress, the State has already appointed a State level Control Board for Cancer and a Working Group for Oral Health. It is also under consideration to appoint sub-committees for Cancer Control, one of which will be for control of tobacco.

Integration with general health

No public health programme will succeed unless it is integrated with the general health services and all the staff right from grass root level of Health Guides, Multipurpose Workers (male/female), Health Assistants, Medical Officers of Primary Health Centres, Rural Hospitals and District Hospitals are involved. It envisages conducting various training programmes for this staff with the involvement and assistance of the Medical and Dental Colleges.

Community participation

Community participation is the key to the success of public health programmes. Therefore, community leaders, teachers, voluntary organisations and even professional bodies will be fully involved in the programme.

Involvement of voluntary organisations

At present, Goa Cancer Society is doing an excellent work in the district of Sindhudurg and Ratnagiri, where they have already held health education camps for teachers in that area and have created very good awareness. Other voluntary organisations are also contributing their share.

School health

For attaining success in the long run preventive programmes should be directed at children. Action is being taken to include the important topics and aspects of various programmes in the health curriculum. It is observed that this not only creates awareness among students, but also students, many a times, persuade their parents to give up these dangerous habits.

Legislation

It is our experience that legislation without proper public awareness and commitment will not be successful. Therefore, the State Government has not

thought of any legislation for the present.

Conclusion

Every State will have a very important role to play in control of tobacco. Unless the State Health Department is convinced of the seriousness and magnitude of the problem and unless a definite State level programme is formulated and its implementation started, it is likely that there cannot be any significant impact on control of tobacco. The State of Maharashtra is committed to this task and is doing its best to take effective steps within the limited resources available at its disposal. The programme can be augmented as well as intensified if regular National Cancer Control and Oral Health Programmes are launched with adequate technical and financial support.

Tobacco and Health : The Indian Scene. L.D. Sanghvi and Perin Notani (Eds.)
UICC Workshop, Tata Memorial Centre, Bombay 400 012, India. (1989)

Action on Tobacco Control : Role of the Consumer Guidance Society

Shalini Sirur[1] and Chitra Sirur[1]

Introduction

In 1982, the U.S. Surgeon General described smoking as the Chief preventable cause of premature ill-health and early death.

In January 1986, the WHO Executive Board, at its meeting, issued the strongest statement they have ever made, on the hazards of tobacco use. "Cigarette smoking is the major cause of ill health and premature mortality in countries where it is widespread".

"Since the evidence of a causal relationship between the use of tobacco, whether for smoking or chewing and ill health is now overwhelming, the onus of proving that tobacco use is not a cause of disease should be on the tobacco industry itself".

Gradually, as the developed countries decreased smoking, the industry started dumping their products in the developing countries where there was virtually no restriction on advertising and no appreciable awareness among the people about the hazards of smoking. Hence the International Organisation of Consumer Unions (IOCU) at its 11th World Congress in Bangkok in December 1984, launched a campaign called AGHAST (Action Groups to Halt Advertising and Sponsorship of Tobacco). This campaign includes the monitoring of promotion and marketing practices of the tobacco industry, the production of action books and lobbying tools in support of local action and international advocacy.

Involvement of Consumer Guidance Society-India (CGSI) in anti-tobacco activities

The Consumer Guidance Society of India is a member of the IOCU Council. The Society decided to take up the issue and launched its GASP Campaign (Group Against Smoking Promotion) in July 1985 and with the aid of the press, campaigned against the promotional tactics adopted by tobacco companies which are aiming at luring young people to take up the smoking habit.

A new brand of cigarettes was being promoted at that time by holding "break dance" shows and free distribution of cigarettes to students and office goers.

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[1] Consumer Guidance Society of India, Hutment J, Mahapalika Marg, Bombay 400 001, India.

Students were being employed for this purpose. Due to CGSI's press campaign, the company immediately withdrew the promotion and the students of the college, where these shows were being held, voluntarily agreed to a ban on smoking on the campus.

In October 1985, two CGSI office bearers attended a Workshop held under the joint auspices of the UICC and IOCU in Penang, Malaysia. The Workshop was on smoking control policies and strategies for developing countries. The Workshop was an eye opener with regard to the extent of the promotional tactics resorted to by the tobacco industry in developing countries. It was also a very valuable guide for developing strategies suited to each country's varying social, economic and political factors that influence the continuing support to a product, that is known to have no safe level of use.

Medical and health organisations are doing their best to educate people about the hazards of tobacco. Voluntary groups like ours also have an obligation to consumers to put the real hard facts about tobacco before them and also to work at all levels to counter the influence of tobacco advertising, especially on young people.

Resolutions adopted at UICC-IOCU Workshop

The thirty one organisations from ten Asian countries adopted the following resolution : 1) to use every opportunity in their countries to inform consumers that tobacco products are highly dangerous 2) to expose the efforts of those with vested interests to lure children and adults to the smoking habit and the pressures they put on governments not to take effective action to deal with the smoking problem.

The groups also decided that they should prevail upon their own governments to 1) recognise that tobacco products are highly dangerous, 2) introduce progressive ban on tobacco uses and 3) initiate large scale and long term health programmes.

Objectives of CGSI

Our own immediate objectives were

1. To campaign strongly against smoking promotion by cigarette companies who are directing their sales pitch towards young people.
2. To educate school and college students about the hazards of smoking by holding workshops and group discussions in colleges as part of our ongoing consumer education programmes in schools and colleges.
3. To interact with other organisations with similar objectives and produce posters, leaflets, anti-tobacco material for distribution.
4. To keep a good rapport with the press and write articles on tobacco and its harmful effects and also build up public opinion against tobacco and its

promotion.

5. Make strong representations to government
   a) To ban sports sponsorship by tobacco industries.
   b) To introduce a ban on advertising.
   c) To make the statutory warning more meaningful and more prominent on packets as well as hoardings and advertisements.
   d) To introduce the internationally recognised danger signal so that the warning reaches even the illiterate consumer of tobacco products including bidis.
   e) To produce audio visuals, anti-tobacco posters, stickers etc.
   f) To carry out surveys and monitor advertising.

Anti-tobacco programmes implemented by CGSI

Two workshops have already been conducted at two colleges in Bombay city. The participation of students was very encouraging. These workshops were held at the H.R. College and Lala Lajpatrai College. The vacations, examinations and tight schedules of the colleges make it difficult to hold more than two workshops in a year.

Survey conducted by the students in the two colleges showed that 75% of the smokers had started smoking around the age of 15 years and 35% of them (around 20 years of age) were already smoking 20 cigarettes a day. Most of them agreed that cigarette advertising is an important influence in their taking up smoking.

There are a few advertising agencies, who on principle, do not accept tobacco and alcohol advertisements. One of them, Sistas Pvt. Ltd., have helped us considerably, in our workshops etc. Mr. S.V. Sista, the Managing Director of this advertising agency addressed the students at the H.R. College Workshop on the subject of tobacco advertising and said that if a product is allowed to be manufactured, advertising of the product cannot be banned. So the focus of anti-smoking campaigns should be on the rights and privileges of the non-smokers. Sistas Pvt. Ltd. produced the posters and leaflets for CGSI's campaign.

Voluntary organisations need audio visuals, educational materials etc for carrying on the anti-smoking campaign and for this funds are always a problem.

With our limited resources we have produced some literature and posters and these have been distributed to many groups who have approached us for help in starting anti-smoking campaigns in their areas. This has mainly been due to the publicity our workshops have generated, not only in newspapers but also in few youth magazines.

The Health Ministry and other concerned Ministries have been receiving letters from us time and again to persuade them to introduce legislations regarding :

1. Display of levels of nicotine and tar content on packets.
2. Specific warnings of health hazards on cigarette packets and advertisements in prominent and bold lettering.
3. Printing of internationally recognised danger signal, (skull and cross bones) on cigarette and bidi packets for the benefit of illiterate consumers. The Health Ministry has decided to use this soon.
4. CGSI has protested against sports sponsorships by tobacco industry.
5. We have also protested strongly about importing of cigarettes. This protest was supported in many newspaper editorials.
6. We have also urged an increase in taxes on cigarettes to discourage consumption.

CGSI has written to Indian Airlines that the "no smoking" section on its Boeing 737 flights should include the first row and not just the 2nd to 7th row as at present. In fact, following the example of many foreign airlines, it might be better to ban smoking during flights totally.

We are happy to note that the government is taking steps against smoking recently by 1) banning of smoking in hospitals, government offices and some public places and 2) showing anti- tobacco slogans on television to discourage the habit.

The recent trend in advertising shows a marked increase in the use of images and situations which would lure young people to this habit. Many advertisements do not display the necessary warning at all, and when they do, it is so blurred and insignificant that CGSI has decided to monitor the advertisements and examine the possibility of taking up such offences seriously with the proper authorities.

A collection of offensive advertisements are also being sent to the Health Ministry to reinforce our plea that advertising of tobacco should be restricted if not banned totally.

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Role of Medical Profession in Tobacco Control Programmes

R.S. Rao[1]

Introduction

"As is your Lifestyle–so is your Cancer" very aptly describes the causation and prevention of cancer. Every society has certain norms of behaviour. These norms are not constant but also change with times. Many lifestyles may have a religious, cultural or historical genesis for a particular country, but has been radically modified by events or lifestyles in other parts of the world.

It has already been clarified that one third of cancer in India are preventable and related to tobacco. Ultimately the society has to change its attitude towards smoking and chewing of tobacco. This is the only permanent solution to the problem. The medical profession has a key role in bringing about this transformation.

The medical profession should lucidly present all scientific data pertaining to tobacco and health. It should guide the advertisers and information media. Secondly it should guide the government for enacting legislative measures. Thirdly, it should set an example to the society by its own behavioural pattern.

Established data on tobacco and health

All the ill-health caused by tobacco should be presented comprehensively. It is immaterial whether the doctor is a cancer specialist or a cardiologist or a family physician; but when presenting data on tobacco, he should take an overview. What is important is that a person should quit smoking. It does not matter, for example, if a woman gives up smoking because of fear of cancer or its ill-effects on pregnancy. The total picture against tobacco should be presented.

We may briefly summarise the ill-effects of tobacco as follows :

1. Tobacco causes cancer of the lip, cheek, tongue, pharynx, larynx and lung. It is also implicated in the causation of cancers of the oesophagus, bladder and cervical cancer. About 200,000 new cases of cancer, registered every year in India, are due to tobacco abuse.
2. It causes chronic bronchitis and emphysema which are crippling, respiratory diseases.

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[1] Tata Memorial Hospital, Dr. Ernest Borges Marg, Bombay–400 012, India.

3. It causes coronary artery disease and is responsible for heart attack and deaths, particularly in young persons.
4. It causes peripheral vascular diseases, gangrene of toes, cramps in muscles etc.
5. It affects the foetus in a pregnant mother who smokes or chews tobacco. The baby tends to be small, and has low birth weight.
6. It produces disturbances of vision and hearing.
7. Peptic ulcers are more prevalent in smokers than in nonsmokers.
8. Smoking and chewing pan with tobacco stains the teeth and nails, and leaves a characteristic odour in the mouth.
9. It leads to very poor oral hygiene.
10. Smoking cuts down the physical stamina of an individual and adversely affects the performance of athletes.

Other known facts regarding tobacco use

1. There is an increased amount of smoking in the West among women. This has resulted in cancer of the lung being the commonest cause of death of all cancer deaths among women in USA. Breast cancer is the commonest cancer, but many are cured, hence not the commonest cause of all cancer death.
2. Passive smokers are those who keep close company with smokers, e.g., wives of smokers, colleagues in office, co-passenger in train or plane. The dangers of passive smoking have been well documented and accepted and in many western countries, their rights are respected e.g. nonsmokers have separate area in the plane. An individual may smoke and ruin his life, but has no moral right to put at risk the life of his fellow human beings, particularly his wife and children.
3. A lot is talked about environmental pollution and the atmosphere being polluted by factory fumes, automobile exhausts and so on. It has been estimated that the inhalation of these pollutants for 24 hours is equal to smoking two cigarettes per day. This has been worked out on the basis of comparing the quantity of carcinogenic substances like benzo(a)pyrine and benz(a)anthracene inhaled in polluted air and from cigarettes.
4. Very relevant information that needs to be highlighted is that bidi smoking is more dangerous than cigarette smoking (1). Table No.1 gives the details of this study. Eighty per cent of smokers in India are bidi smokers. There is no quality control on bidi manufacture nor any warning sign on the bidi packet. The popular myth is that four bidis are equivalent to one cigarette. Bidi is also responsible for cancer in the oropharynx and upper respiratory tract.
5. Harmful effects of chewing tobacco : This habit is largely indigenous to the Indian sub-continent. Tobacco is chewed with pan leaf, lime and various ingredients. Some of the ingredients produce excessive salivation. The tobacco

Table 1. Chemical analytical data for a bidi and a US cigarette[1]

Parameters	Bidi (2 puffs/min)	US cigarette (2 puffs/min)	US cigarette (1 puff/min)
Weight, mg	573	1150	1150
Length, mm	76	85	85
Draw resistance, cm	10.4	7.8	7.8
Neophytadiene, % of dry tobacco	0.036	0.28	0.28
Burning rate, mg/min.	–[2]	59.0	59.0
Puffs	23.1	16.1	10.8
Mainstream smoke			
pH Puff 3	5.9	6.09	5.94
5	5.8	6.04	5.93
7	5.7	5.90	5.91
9	5.7	5.84	5.86
last	(23) 5.67	(16) 5.84	(10) 5.86
A. Gaseous phase			
Carbon monoxide, vol. %	7.7	3.51	3.76
Carbon dioxide, vol. %	9.4	8.71	8.77
Ammonia, μg	284	180	79.6
Hydrogen cyanide, μg	903	445	240
Isoprene, μg	533	934	582
Acetaldehyde, μg	751	1110	770
Acrolein, μg	67	129	83.5
B. Particulate phase			
"Tar" wet, mg	51.4	54.2	31.5
"Tar" dry, mg	44.9	50.7	27.9
Nicotine, mg	1.71	3.0	1.78
"Tar" FTC[3]	43.2	47.7	26.1
Phenol,μg	249	148	86.4
o-Cresol,μg	55.3	34.4	20.4
m- and *p*-Cresol,μg	139	89.5	49.5
2,4-Dimethylphenol,μg	27.6	17.4	9.0
p-Ethylphenol,μg	41.6	32.1	18.2
Benz[a]anthracene, ng	117	81.3	44.4
Benzo[a]pyrene, ng	78	46.7	25.1

[1] The analytical deviations were ± 5% for the cigarettes and ± 8% for the bidis with the exception of benz[a]anthracene and benzo[a]pyrene which were ± 8% and ± 10% respectively.

[2] Only limited free burning; insufficient for calculating burning rate.

[3] "Tar" FTC—Tar minus water minus nicotine.

Source : Ref. 1.

chewers usually do not swallow the pan juice with tobacco and retain it in the hypopharynx. Other ingredients cause an astringent action. A dry bolus is also kept in the cheek. These habits explain the different patterns of oral and pharyngeal cancers seen in different parts of the country.

6. Tobacco tooth paste : Another danger is the presence of tobacco in some of the popular brands of tooth paste. A lot of advertisements go into the promotion of these brands of tooth paste and goes unchallenged.

To convey the message to the lay public

It is essential that having defined the message, the medical profession should take the help of professionals in advertising agencies to convey the message to the public in a manner which will be acceptable to them. This is an area of speciality by itself. It is important to know the public mood, its tastes, its likely reactions etc. The message should not be repetitive, boring or put off the reader or viewer completely. For example some of the recent advertisements on family planning which have appeared on the Indian television screens are well planned, appealing, not vulgar and are varied. Similar effort should be made against tobacco.

Role of medical profession in bringing about legislation

Certain areas need urgent attention of the governments and the politicians e.g. the harmful effects of bidi and some control of bidi industry, improvement in the sense of public hygiene, and law against spitting. It has been postulated that the incidence of oral cancer in Indian community in Singapore has gone down because of stringent enforcement of laws on spitting in public places.

The Government should be appraised of these facts and pressurised to take suitable action. It is worthwhile raising a public controversy on this issue and invite a public debate. Leading men of medical profession should speak out on dangers of bidi smoking. A heightened degree of public awareness of dangers of bidi smoking should be created before government can enact any laws.

The Maharashtra Government has banned smoking in hospitals, secretariat, courts etc. and has strictly enforced this law. This indeed is a welcome step.

We would urge the Government to ban chewing of pan when on duty and make indiscriminate spitting in public places an offence. I am sure this will go a long way in preventing oral cancer.

We should also advocate strong measures against the use of tobacco in tooth pastes.

Exemplary behaviour of doctors

Doctors, be they in the village or in the city, are peers of society and are trend setters in matters of health. Therefore, it behoves on us to conduct ourselves in an exemplary manner. We should not smoke or chew pan in our dispensaries, clinics or in public functions. It should preferably be avoided even in homes and private

parties. Even in such small groups, people observe us keenly, particularly the younger and impressionable persons. How we speak, how we talk, how we conduct ourselves are noticed and often adopted by them as behaviour.

To help people give up tobacco habit

Tobacco is an addiction. Many persons would like to give up smoking and chewing tobacco, but are unable to do so or revert back after a period of abstinence. They need understanding and help from the medical profession to get over the addiction. All our professional skills should be brought to focus on this problem without looking down on the patient or giving him a long sermon.

To help people give up pan chewing habit

Not much attention has been given to help the pan chewers.The unhygienic aspects of pan habit should be stressed to young people. The importance and appeal of white shining teeth and repulsiveness of bad breath should be stressed. Those who chew tobacco and are unable to give up should be asked to brush teeth last thing at night and not to keep tobacco in the mouth for long hours at night. Just as we advise smokers to have chest x-ray once a year, we should advise pan chewers to go to a dentist or doctor once a year to have his oral cavity examined.

Conclusion

In conclusion, the medical profession has an important role to play in tobacco control programmes. It should define the scientific data on harmful effects of tobacco in a lucid manner. Once the message has been defined, it should be left to the professionals in advertising media to convey it to the public. It should guide and influence the Government to enact legislation against tobacco. The doctors should conduct themselves in an exemplary fashion. They should help persons who wish to give up the addiction. They should insist on an annual medical check up of oral cavity among tobacco chewers and a chest x-ray for smokers.

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Tobacco and Health : The Indian Scene. L.D. Sanghvi and Perin Notani (Eds.)
UICC Workshop, Tata Memorial Centre, Bombay 400 012, India. (1989)

Action on Tobacco Control : Role of National Service Scheme

Babu Mathew[1]

Introduction

The National Service Scheme (N.S.S.) is a voluntary organisation for college students, which was started in 1960s at the instance of the late Prime Minister Pandit Jawaharlal Nehru. It mainly aims at exposing the college students to the village life and rural society. Each student is enrolled in it for a period of two years. During this period, the students are engaged in various humanitarian and social activities, the most important being a ten day camp in an adopted village. In the village, they may be engaged in the following types of work.

1. Educational programmes
2. Health programmes
3. Agricultural programmes
4. Self employment programmes
5. Recreational programmes

Each state has a Programme Advisor. Under him, each University has a Programme Co-ordinator. Each college may have one or two units with an average strength of about 120 student volunteers per unit. Each Unit is under the control of a Programme Officer who is a regular staff member of the college. Depending on the type of training the students receive in the college and considering the general needs of the locality, the units may undertake one or more activities for implementation. They meet often at the college or at work-locations. A certificate is given to each volunteer on the successful completion of his term.

At present in India, there are about 7 lakh (0.7 million) N.S.S. volunteers and by the end of the 7th plan, the number may be raised to one million. In Kerala there are 316 units spread over the Universities of Kerala, Gandhiji and Calicut. The volunteer strength is over 37,000. By the end of 7th plan the volunteer strength may go up to 40,000. The activities of each unit is monitored by the Programme Co-ordinator from time to time and a special committee inspects each unit and evaluates the work. Best unit in each University is awarded prizes annually.

Utilization of N.S.S. volunteers (Kerala State) in the fight against cancer

In January 1986, a workshop was conducted for N.S.S. Programme Co-ordinators

[1] Community Oncology Division, Regional Cancer Centre, Trivandrum, India.

of Southern States at the Kidwai Memorial Institute of Oncology, Bangalore. During this workshop it was decided to utilize the "Youth Power" of the N.S.S. for prevention and control of cancer. The main areas of action were decided to be (1) health education, (2) conducting cancer detection camps and (3) various modes of assistance to patients where cancer hospitals exist.

Phase I of the project

A plan to implement these activities in the State of Kerala was prepared by the Community Oncology Division, Regional Cancer Centre, Trivandrum. According to this the State is to have a three tier organisation to implement a two phase programme by each unit of the N.S.S.

Tier one of the organisation

The first tier is a Central Unit composed of all Programme Co-ordinators of the State, and a few doctors of the Regional Cancer Centre and other Medical Colleges as resource persons. This unit was to plan and monitor all activities of the State, train the Trainer Units, generate information and produce software for use of all units.

A book showing the various aspects of cancer prevention to be used for public education was printed in August, 1986. Chapter 2 of this book gives in detail the information that every volunteer should know about smoking and chewing of tobacco. It also gives information about the advantages of quitting the tobacco habit. Posters were developed for health education, of which, ten were specifically against tobacco use.

Tier two of the organization

The second tier is the "Trainer Units". There are five Trainer Units based in the five Medical Colleges of the State. These Trainer Units are composed of the Programme Co-ordinator of the University under whom the college is placed and a few doctors of the Medical College, a few selected senior student volunteers of the Medical College and Programme Officers of other colleges around the locality. Their main activity is to train all the units in their jurisdiction in implementing activities for the prevention and control of cancer. Each of the Trainer Units was given training in their locality by the Central Unit in a one day workshop held during the months of August and September and 426 persons attended these programmes.

Tier three of the organization

The third tier is a "Peripheral Unit" located in each unit of N.S.S. It is composed of the programme officer of the college, one medical practitioner of the locality as the resource person, the student volunteers of the Unit and other interested staff members of the college.

The Peripheral Unit is to implement a two-phased action on tobacco control as part of their activities on prevention and control of cancer. The first phase was to arrange a meeting in each college on 2nd October 1986. In this meeting two or three resource persons spoke on the various aspects of smoking, alcoholism, and drug addiction. At the end of the meeting all the volunteers took oath to abstain from tobacco and keep N.S.S. a non-smoking society. Over 300 units in the State participated in this phase of the anti-tobacco activity.

A poster and slogan competition was also conducted, the theme being "Harmful effects of tobacco".

Phase II of the project

The phase-two activity was taken up during the 10-day camp in the village area by those units who had health education as one of their activities. Such units were trained in health education by one of the trainer units prior to their camp. Students organised public meetings in the villages especially inviting the school teachers, grass root level workers of the Health, the Community Development and the Agricultural Departments, *Angan-Wadi* (creche) workers, members of Youth Clubs, and *Mahila Mandals* (women's organisations) of the village. Two or three students and one resource person (member of the Central Unit or Trainer Unit) spoke on the harmful effects of tobacco. The meetings were held in the late evenings and were followed by a film or video show on tobacco related diseases. Posters developed by students and Central Units were exhibited at the venue of the meetings. The Peripheral Units extracted information from the printed booklet and reproduced it to suit the locality and distributed the material throughout the village after the health education classes. Thirty six colleges had completed the phase-two activity by 31st March, 1987.

Conclusion

This pilot project proposed and executed in Kerala for the first time, has proved to be feasible and can be implemented without any additional budget allotments. Based on our experience, the materials used for training and health education can be modified to suit the need of each State. The strategies for communication, may also be modified to fit with the culture and beliefs of the people of each State.

Appendix

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Possible Substitutes and Alternate Uses of Bidi Tobacco

B.G. Jaisani[1]

Introduction

In tobacco map of the world, India occupies second position next only to China in area and third position in total production of tobacco. India accounts for about 10.8 per cent of the world tobacco area and 8.4 per cent of the world tobacco production (Table 1).

Table 1. World tobacco–area and production (1983-84)

Country	Area '000 ha	Production '000 MT	Productivity kg/ha
China	748.0	1400.0	1872
India	440.1	497.1	1130
USA	321.9	746.0	2317
Brazil	273.0	373.0	1366
Turkey	208.0	210.0	1010
World Total	4078.0	5920.1	1452

Tobacco industry in India

Home consumption

Tobacco an important cash crop of India occupies 0.3 per cent of the total cropped area of the country. Among excise earning agricultural commodities, it earns highest revenue which was Rs. 1,070 crores during 1983-84. This was accrued from 410 million kg. of tobacco used by the domestic market (Table 2).

Export

In international tobacco trade, India ranks sixth after USA, Brazil, Zimbabwe,

[1] All India Co-ordinated Research Project on Tobacco of Indian Council of Agricultural Research. Gujarat Agricultural University, Anand Campus, Anand–388 110, India.

Greece and Turky. In case of Virginia flue-cured tobacco India's share is about 10.8 per cent and ranks fourth after USA, Brazil and Zimbabwe. The principal markets for unmanufactured tobacco from India are UK, USSR and Japan. India also exports manufactured tobacco. The export earnings during 1983-84 was Rs. 204.6 crores (Table 3)

Table 2. Quantity of tobacco cleared for home consumption 1983-84.

Type	Quantity (million kg.)
Flue-cured virginia	57.5
Other cigarette types	34.5
Bidi	143.0
Cigar and cheroot	9.0
Snuff	4.0
Hookah	17.5
Chewing	35.0
Misc. purposes	110.0
Total	410.5

Table 3. Export of tobacco and tobacco products from India variety-wise (1983-84)

Tobacco Type	Quantity '000 kg	Value '000 Rs.
1. Unmanufactured	89,985	18,10,523
2. Manufactured		
– Bidis	867	41,919
– Cigarettes	1,842	86,546
– Chewing	59	11,635
– Hookah paste	14,105	91,331
– Snuff	36	2,009
– Zarda scented	17	1,685
– Others (cigar)	8	638
Total manufactured	16,934	2,35,763
Total of 1 & 2	1,06,919	20,46,286

Employment generation

On employment front, tobacco is a labour intensive crop and provides

employment to millions of people in its cultivation, curing, grading, redrying, packaging, manufacturing and marketing. About7.5 lakh growers and curers are engaged in cigarette tobacco production. About 5.5 lakh people are employed in cigarette industry. Whereas bidi industry employed about 30 lakh people which is more than double the number employed in cigarette production. Furthermore mostly tribals and rural poor are engaged in rolling bidis and collection of tendu leaves from the forests. Thus, bidi industry has helped in preventing influx of rural labourers to urban centres. Besides cigarette and bidi industries, snuff industry also provides livelihood to several thousands of people.

Total area and distribution of tobacco in different states of India

In India, tobacco is cultivated in varied agroclimatic conditions. The principal tobacco growing states are Andhra Pradesh, Gujarat, Karnataka, Bihar, West Bengal, Orissa, Tamil Nadu, Maharashtra and Uttar Pradesh. The various types of tobacco grown in the country are flue-cured (cigarette), bidi, hookah and chewing, natu, lanka, cheroot, cigar filler as well as wrapper, burley and *rustica*. Among different types, the production and the productivity of bidi tobacco is the highest (Table 4).

Table 4. Area and production of different types of tobacco in India (1983-84)

Type	Area '000 ha	Production 1000 MT	Productivity kg/ha
Virginia	149.8	135.2	903
Natu	43.0	56.0	1302
Bidi	126.0	175.5	1393
Cigar and cheroot	14.2	15.0	1056
Hookah	31.4	33.8	1076
Chewing	69.7	73.9	1060
Snuff	6.0	7.7	1283
Total	440.1	497.1	1130

Among different states, Gujarat state occupies second place next only to Andhra Pradesh in area and production. Gujarat shared 25 per cent of the area and 38 per cent of the total tobacco production in the country. However, from productivity viewpoint, Gujarat stands first among all the tobacco growing states (Table 5).

Bidi tobacco

Bidi tobacco is mainly grown in the state of Gujarat. Among different types of tobacco grown in Gujarat, bidi tobacco accounts for 85 per cent of the total

Table 5. Area, production and productivity of tobacco in different states (1983-84)

State	Area '000 ha	Production '000 MT	Productivity kg/ha
Andhra Pradesh	187.1	191.9	1026
Bihar	13.4	13.4	1000
Gujarat	108.3	186.9	1726
Karnataka	48.0	28.8	600
Maharashtra	10.1	7.9	782
Orissa	18.6	10.2	548
Tamil Nadu	12.4	13.7	1105
Uttar Pradesh	14.7	19.7	1340
West Bengal	15.4	15.5	1026
Others	12.4	9.8	790
All India	440.1	497.1	1130

production. District-wise data on area and production suggest that Kheda and Vadodara are the major tobacco growing areas in Gujarat. Bidi tobacco is also grown in about 33,000 hectares in Nipani area comprising Belgaum district of Karnataka and parts of Kolhapur and Sangli districts of Maharashtra state.

Bidi tobacco occupies 53.6 and 35.9 per cent of total area respectively in those

Table 6. Percentage area covered by tobacco vis-a-vis other crops in the area having tobacco-based farming system (1984-85) in the states of Gujarat and Karnataka

Crop	Gujarat	Karnataka
Food crops	33.7	32.2
Groundnut	3.7	17.6
Castor	0.8	–
Tobacco (bidi)	53.6	35.9
Cotton	2.1	–
Sugarcane	–	8.2
Fruit crops	1.7	–
Vegetables	0.7	0.9
Fodder crops	2.0	2.9

regions of Gujarat and Karnataka which have tobacco-based farming system (Table 6) next in order being food crops in both the regions, suggesting that tobacco is grown in a concentrated area.

Cost of cultivation of tobacco

The survey undertaken to determine the yield levels, cost of production and net returns in the Middle Gujarat as well as Karnataka revealed that the average net return of the farms irrespective over holdings was Rs. 5667.20 per hectare for Gujarat and Rs. 3190.90 per hectare for Karnataka (for bidi tobacco). The net return for chewing and *rustica* tobaccos were Rs. 3052.90 and Rs. 3305.90 per hectare respectively/ in Gujarat (Table 7).

Table 7. Cost of cultivation of bidi tobacco in Gujarat and Nipani areas (1984-85)

Category	Gujarat			Nipani areas		
	Yield kg/ha	Cost of production Rs./ha	Net realization Rs./ha	Yield kg/ha	Cost of production Rs./ha	Net realization Rs./ha
Marginal	1830	8488.1	2913.5	–	–	–
Small	1916	8747.0	3621.8	1093	6975.8	2785.1
Medium	1885	9255.7	4020.1	1116	7373.1	3056.6
Large	2107	10005.4	6709.3	1226	8424.7	3547.2
All farms	2033	9636.9	5667.2	1155	7693.4	3190.9
Chewing tobacco	2112	11487.8	3052.9	–	–	–
Rustica tobacco	2265	9081.5	3305.9	–	–	–

Possible substitutes of tobacco by other crops (Gujarat State)

Comparison of net realization of tobacco made with other crops grown in Middle Gujarat revealed that irrigated castor fetched the highest net returns of Rs. 9,702 per hectare followed by irrigated cotton which realized Rs. 9,560 per hectare. However, in the irrigated tobacco fields two crops viz., tobacco-summer *bajra* (a millet) or tobacco-summer groundnut could conveniently be taken with a net realization of approximately Rs. 5,500 for tobacco-summer *bajra* and Rs. 5,700 for tobacco-summer groundnut per hectare (Table 8). As regards the cropping sequences, tobacco-tobacco is the normal crop sequence in majority of the tobacco growing areas. Under assured water supply, tobacco-summer *bajra* is the normal crop rotation. However, crop rotation studies have indicated that

tobacco-summer *bajra* (first year), cotton (second year) and tobacco-summer *bajra* (third year), rotation gave more remuneration than the traditional crop rotation of tobacco – tobacco. Tobacco-summer groundnut is also becoming a popular crop sequence in the tobacco growing areas of Gujarat.

Table 8. Economics of tobacco vis-a-vis other crops in Gujarat State

Crop	Yield (kg/ha)	Cost (Rs./ha)	Gross realization (Rs./ha)	Net realization (Rs./ha)
Tobacco	2093	5575	9942	3367
Bajra (Summer)	3025	3835	6050	2215
Cotton	2740	5510	15070	9560
Groundnut (Summer)	1327	4170	6635	2465
Soyabean	772	2165	2316	151
Castor	3575	2810	12512	9702
Tur (Rabi)	403	2890	1612	-1278
Sesamum (Summer)	190	2275	1140	-1135

Constraints in implementing the possible substitutes

The substitute of tobacco with more remunerative crops like cotton, castor, sugarcane and groundnut in tobacco growing areas is likely to pose some problems. Firstly, farmers are growing tobacco due to the problems of stray cattle and theft to which tobacco is practically immune. Added to it is the fragmentation of holdings which again adds to the problem of security. Secondly, infrastructural facilities created for the processing of tobacco, which are specialized in its nature cannot be used for any other crops. Pesticide residue problems in ecosystem will be aggravated as other commercial and food grain crops require very heavy use of pesticide umbrella. Lastly no other crop is as drought tolerant as tobacco, hence it is really difficult to find a substitute in rainfed agriculture. Last and not the least is that millions of tribals and village poors will become jobless as large population is engaged in rolling bidis during off seasons and tribals' livelihood is largely dependent on the tendu leaf collection.

Alternate uses of bidi tobacco crop

Tobacco is a rich source of several phytochemicals viz., nicotine, nicotine acid, nicotine amide, solanesol, organic acids (malic, oxalic and citric), leaf protein

concentrate (LPC) and pentosans.

Tobacco industry throws out a large tonnage of waste material,approximately 10 percent of its annual production. This is excluding the stalk for which exact statistics are not available and is presumably burnt as fuel. Of the approximate 45 million kg tobacco wastes, 21.5 million kg is available from Andhra Pradesh, 16.2 million kg from Gujarat, 2.7 million kg from Karnataka, 2.1 million kg from Tamil Nadu and 1 million kg from Maharashtra. Out of these, 20 million kg of wastes available from Gujarat and Nipani (parts of Karnataka and Maharashtra states) is a potential source for the manufacture of insecticide grade nicotine sulphate (40%), which acts as a stomach, contact and fumigant poison.

Table 9. Production potential of some of the phytochemicals from bidi tobacco waste (Raw + Dust)

Constituent	Per cent	Estimated production potential (tonnes)
Nicotine	2.39	326
Organic acids		1358
Malic	4.55	
Citric	1.30	
Oxalic	4.14	
Solanesol	0.50	68

The production potential of different phytochemicals from bidi tobacco wastes works out to be 326 tonnes of nicotine, 1,358 tonnes of crude organic acids and 68 tonnes of solanesol (Table 9). The export figures of nicotine sulphate indicate that 346 tonnes of nicotine sulphate was exported to Japan, U.K. and Canada during 1981-82 (Table 10). However, intelligent market survey indicates that about 800 tonnes of nicotine sulphate was exported to Japan alone during 1985-86. The cost of production of nicotine sulphate works out to be around Rs. 35.45 per kg while its selling price ranges from Rs. 65-90 per kg of nicotine sulphate*.

The nicotine sulphate exported to Japan is mainly used as insecticide on orchards. If local markets are developed for the use of nicotine sulphate on orchards and vegetables to avoid the problem of pesticide residues, the demand for nicotine sulphate may increase at a phenomenal rate. To cope up with this demand, the main crop could directly be used for the production of nicotine

* Personal Communication with M/s. Nicosulph Industries Pvt. Ltd., Dakore, Dist. Kheda, Gujarat.

sulphate by giving remunerative prices to the growers. Patel and Ramakrishnayya (1975)[+] have indicated that it would not be economical to use the main crop for the production of nicotine sulphate in comparison to the wastes' utilization. But the recently released bidi tobacco variety "Gujarat Tobacco 5", which is a nicotine rich variety and has very high yielding ability than the existing varieties (Anand 119 and Anand 2), could be considered for utilization of nicotine extraction. All parts of it are richer in nicotine content than Anand 119 (Table 11).

Table 10. Export of nicotine sulphate (tonnes) from India

Country	Year			
	1978-79	1979-80	1980-81	1981-82
Japan	241.8	312.2	262.9	337.0
U.K.	9.7	3.1	2.1	8.1
U.S.A.	12.1	5.1	6.9	–
Belgium	–	–	1.9	–
Canada	–	0.9	–	0.9
Total	263.6	321.3	273.8	346.0

Table 11. Nicotine content of different plant parts of bidi tobacco

Plant part	Variety	
	A 119	G.T. 5
Lamina	7.01	9.51
Midrib	0.58	1.60
Stalk	0.63	1.32
Root	0.72	0.95
Ratoon crop	1.66	3.50

Forgoing proposition can thrive better if we really make an alternate use of tobacco by developing an integrated process for the extraction of insecticide grade nicotine sulphate, pharmaceutical grades organic acids and solanesol, food grade

[+] Patel GJ, Ramkrishnayya BV : Tob Res 1(2) : 110–115, 1975.

tobacco leaf protein and pentosans for the production of ferfural, an industrial solvent, by making use of 90 day old immature crop of tobacco (Table 12). Besides this, tobacco can be exploited as an oil seed crop, which gives the seed yield of about 1,000 kg/ha having 40-42 per cent oil content. The leaf available from such crop could be utilized by nicotine sulphate industry.

Table 12. Phytochemicals in immature crop of tobacco

Per cent constituent	Tobacco waste	90 day old crop		
		Plant parts		
		Lamina	Midrib	Stalk
Nicotine	2.39	3.58	0.85	0.50
Organic acids	9.99	10.43	ND	5.75
Solanesol	0.50	0.50	–	ND
Pentosans	–	ND	8.41	13.39
Protein *	–	2.49	0.37	ND

* On green basis

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